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## PNEUMONOCONIOSIS: A SUMMARY OF PRESENT KNOWLEDGE.[1]

By E. L. COLLIS, M.A., M.D., M.R.C.P.,
*Mansel Talbot Professor of Preventive Medicine, The Welsh National School of Medicine, Cardiff.*

THE proceedings of the British Royal Commission on Metalliferous Mines and Quarries, published in 1914, make a convenient starting point, since the evidence gathered by that Commission provided material for the first definite effort at distinguishing various dusts from one another according to the damage they cause; at distinguishing between organic and inorganic dust and between the chemical compositions of different inorganic dusts. The evidence and the Report of the Commission, owing to the immediate intervention of the European War, have hardly received the attention they merit. Briefly put, organic dusts are held to be innocuous, while other dusts are considered to be harmful in proportion as their composition differs from that of the human body; in particular dust of uncombined silica is pronounced to be the most harmful of all. Further, the inhalation of different dusts is recognized to be capable of causing different types of pulmonary disease.

### Definition.

A definition must be arrived at as to what is meant by the term "pneumonoconiosis." Zenker invented the word some sixty years ago to cover pathological conditions of the lungs resulting from inhaling dust. So long as microbes and moulds on the one side and dusts of toxic material, such as lead and arsenic, on the other, are not included under the heading of "dust," the original meaning may still be accepted.

Certain clinical manifestations are commonly accepted to result from dust inhalation and are grouped together as pneumonoconioses without regard to the presence or absence of any pathological condition distinctly due to dust inhalation. Reliance

[1] Read at the Fourth International Congress on Industrial Accidents and Diseases held at Amsterdam, September, 1925.

is placed on statistical deductions, usually based on occupational records, for attributing these clinical manifestations to dust. The diseases usually included are asthma, bronchitis, pneumonia and fibroid phthisis (with or without tuberculous infection).

No pathological condition has been described which distinguishes asthma, bronchitis and pneumonia, when resulting from dust inhalation, from the same diseases as they occur among those not exposed to any special dust risk; that is to say, the pathological conditions usual to these three diseases are present whether their instance is determined by dust inhalation or by other influences. Herein the pathology of these diseases differs from that of phthisis as determined by dust inhalation.

### Asthma.

True spasmodic asthma undoubtedly results from exposure to certain dusts; examples are, dust generated in flax heckling and in the initial processes of cotton spinning, *exempli gratia* in the blow rooms and at the carding engines. The clinical picture was first carefully drawn by Greenhow sixty years ago for flax hecklers. Its distinctive features are that the asthmatic condition is brought on from exposure to dust and that those who have been exposed to it longest, suffer most severely. No protection is acquired from prolonged exposure to this or any other kind of dust; herein exposure to dust inhalation generally differs from many other occupational risks; for instance, a newly employed worker may succumb to an amount of lead dust or of trinitrotoluene which a seasoned worker will withstand, while even industrial accidents are most prevalent among the newly employed. A cotton stripper who at first is only affected during the early hours of Monday morning, becomes, after the passage of years affected for a period extending further and further into the week, until finally he is compelled to stop work. By this time he presents a typical picture of a chronic asthmatic.

Asthma of this type differs from the shortness of breath experienced in advanced cases of pulmonary fibrosis. It is a true spasmodic asthma, brought on by exposure to the causative dust; the extraordinary muscles of respiration are brought into play and in a case of old standing typically square shoulders are present. In advanced fibrosis the shortness of breath results not upon exposure to dust, but upon exertion; the diaphragm is brought into action and the upper part of the chest does not expand; it is a case of air hunger.

Exactly what it is in the dust that gives rise to the condition has not been determined. Recognition of its cause through steps taken to diminish exposure to the dust has led almost to the elimination of the disease. Its importance lies in exhibiting a definite clinical condition which, although associated with some other pneumonoconioses, is entirely distinct from fibrosis. It is a condition which incapacitates, but does not kill; for asthma seldom appears as a cause of death. Nevertheless mortality among those exposed to these dusts would appear to be unduly high from bronchitis—150 for cotton strippers and grinders in 1910-12 as compared with 57 for all cotton operatives; to be high for pneumonia—113 as compared with 76; and somewhat in excess of phthisis—155 as compared with 120. Such statistics indicate that the asthmatic condition is associated with damage to the respiratory apparatus and in particular to the bronchial mucosa.

### Bronchitis.

Bronchitis when caused by dust is in no way different from bronchitis in general, except that it appears rather earlier in life and is more than usually prevalent among those exposed to risk. It causes an excessive amount of ill health and finally contributes unduly to mortality records; as a cause of incapacity it ranks easily first among the pneumonoconioses. Even among groups of workers who experience a greater mortality from fibroid phthisis than from bronchitis, the disease during life leads to far more illness and lost time.

Too little recognition is accorded to these facts, possibly because dust inhalation is by no means the only predisposing cause of bronchitis and the pathological condition which results, does not differ from that of bronchitis due to other causes. Nevertheless whatever be the dust, if it causes trouble at all, it causes bronchitis. No one of the other pneumonoconioses ever occurs without being associated with bronchitis. If any form of lung trouble be suspected of having a dust origin, but bronchitis be not in excess in the group exposed to the dust, then the probability is great that the dust is not to blame. The importance of this fact when examining statistical records is considerable; it is inclined to be overlooked. On the other hand, bronchitis due to dust is usually associated with an undue prevalence of pneumonia; while it is also associated in the case of some dusts with the occurrence of asthma and of other dusts with fibroid phthisis. It seldom, if ever, is the only form of pneumonoconiosis present.

Inhaled dust first comes in contact with the air passages, only small particles are carried on into the alveoli; any sized particle which can be inspired, may reach the bronchi. Probably any form of dust, if inhaled in sufficient amount over sufficiently long periods, is capable of originating bronchitis; but in practice exposure to dusts of animal origin, such as leather and of vegetable origin, such as flour, are not found to cause bronchitis. Here an exception occurs in dust of vegetable husk; but such dust as it occurs in industries is frequently contaminated with inorganic matter, while some vegetable husks even contain silica. Among inorganic dusts those of the various salts of calcium, such as limestone, plaster of Paris and alabaster, do not normally cause bronchitis, nor do those composed of silicates, such as cement and clay. On the other hand, many inorganic dusts, such as those of emery, glass, asbestos, carborundum and silica (in its various forms of quartz, flint and quartzite) may be

expected after prolonged exposure to give rise to bronchitis.

The cause of the trouble is for the most part mechanical. The duty of the ciliated epithelium of the bronchial mucosa is to expel dust particles by entangling them in mucus and then propelling them up the air passages; but if this epithelium is consistently given too much to do, the cells deteriorate and the mucosa becomes chronically inflamed—*id est* a condition of chronic bronchitis is established. In this way the defensive mechanism which protects the alveoli from the intrusion of dust, is damaged.

### Pneumonia.

The seat of election for pneumonia is the finer bronchioles and alveoli. Particles of dust, in order to get so far into the air passages, must be small, probably no larger than those concerned in silicosis, *id est* from one to five microns. After allowance has been made for this question of size, the position, as just summarized with regard to bronchitis, applies equally to pneumonia when it occurs among those exposed to dust. But the resulting ill health and mortality, considerable though they are, fall short of those due to bronchitis.

The same kinds of dust, if sufficiently small, that predispose to bronchitis, predispose to pneumonia; and their influence also appears to be exerted mechanically through interference with the normal activity of the cells lining the air passages. It is a case of mass action, rather than chemical activity; and no pathological condition specific to dust inhalation results. Nature's efforts at removing the inhaled dust result in exudation and congestion of the vessels in the alveolar walls; thereby a nidus is provided for pneumonic infections.

Other influences, such as exposure to inclement weather, to work before hot furnaces and addiction to alcohol, affect the prevalence of pneumonia so much, that dust as a predisposing cause, is in danger of being overlooked. But its importance is considerable and should be remembered, particularly when dust inhalation experiments are being carried out with animals.

### Dust Phthisis.

Advance in knowledge during recent years has been most pronounced with regard to dust phthisis; and the attention paid to this specific dust disease has led to it being regarded (somewhat mistakenly) as the chief of the pneumonoconioses. Certainly in many ways it is the most interesting.

The path of advance was first indicated by statistical research. Thereby was brought to light the now accepted fact that of all dusts the dust of silica is the most harmful; it not only paves the way for bronchitis and pneumonia, but possesses a peculiar and unique power of leading to a specific pathological condition, now known as silicosis, which in its turn predisposes its possessor to tuberculous infection.

The evidence is based on observations that silica and only silica comprises or is a constituent of every dust, concerning which vital statistics display those exposed to risk as experiencing a high death rate from phthisis associated with high death rates from bronchitis and pneumonia; and further that the high death rate from phthisis occurs rather later in life than is usual for this disease.

Consideration of the facts gave rise to the suggestion that silica, quite apart from any mechanical effect it might exert upon the air passages, must when carried from the alveoli into the pulmonary tissues, react chemically with those tissues.

All subsequent statistical inquiries have fully justified the original announcement; hence there is no need to dwell upon them. The interest today rather lies in research undertaken to determine what takes place when silica is introduced into the body. Any attempt to discuss at length all that has been done—and the work is still in progress—would extend this summary unduly. I prefer to err on the side of brevity and to state shortly the facts so far brought to light by different workers whose chief publications are stated in the list of references. They are as follows:

(1) Dust particles which have reached the alveoli are there engulfed by macrophage cells originating from the epithelium lining the alveoli and are carried within them through the pseudostomata into the lymph channels of the lungs.

(2) Particles of some kinds of dust are more readily engulfed than those of others. Organic particles, such as coal, are most readily taken up and particles of silica least readily; but when macrophage cells have been, as it were, educated in the function of absorbing dust particles through the presence of, say, coal dust, they more readily take up particles of such things as emery, carborundum and silica.

(3) Particles of certain dusts, such as carborundum, when engulfed and carried into the lung tissue, remain inert and provoke no reaction.

(4) But silica particles slowly dissolve within and react chemically upon the cells containing them, causing cell death and mummification.

(5) The dead mummified cells collect in and block up the lymph passages and glands.

(6) The final result of the reaction is the formation of fibrous tissue as a solid mass of material which in time replaces the normal alveolar structure of the lungs; the condition is one of silicosis.

(7) All the various stages through which silicosis advances can be detected by X ray photography.

(8) The same results follow whether fine silica dust be artificially placed in the body or soluble silica be injected; but in the latter case the necrotic changes take place more rapidly.

(9) Necrotic tissue so caused forms a nidus in which tubercle bacilli readily flourish. Necrotic tissue caused by other agents does not form so favourable a nidus.

(10) When soluble silica is introduced into the blood stream, it reacts with liver cells causing fibrosis there and also with kidney cells (by which its excretion is attempted) causing fibrosis there.

(11) Statistical records indicate that Bright's disease as a cause of death is unusually prevalent among certain occupational groups exposed to silica dust.

(12) Clinical observations have shown that:

(i.) In any persons who have been exposed to silica dust for a period sufficiently long to enable the dust to gain access to the lungs, the silicotic process continues to advance after complete withdrawal from exposure;

(ii.) the silicotic process may progress to a fatal termination without help from any intercurrent disease; but

(iii.) a silicotic patient is particularly liable to fall a victim to tuberculous infection;

(iv.) tuberculous infection in a lung containing silica particles, hurries on the process of silicosis;

(v.) pulmonary tuberculosis in silicotic subjects, having regard to the amount of fibrosis present, runs an unusually rapid and fatal course; but

(vi.) when silicotic persons are protected from exposure to tuberculous infection, the occurrence of tuberculosis among them is minimized. Hence tuberculosis in such persons appears to result from fresh infection during adult life;

(vii.) tuberculous infection from silicotic persons seems to be less potent than usual for infecting non-silicotic persons.

(13) Statistical inquiry has shown, how, by a method of differential diagnosis, the environmental influence determining an undue prevalence of phthisis, may be detected; thus:

(i.) When the influence is silica dust, the high mortality from phthisis is at its maximum in late middle life and is associated with an excessive mortality from bronchitis and with high mortalities from pneumonia and Bright's disease, but not necessarily from any other cause of death;

(ii.) When the influence is the alcoholic habit, the high mortality from phthisis has its maximum incidence rather earlier in life than for the standard population and is associated with an excessive mortality from diseases attributed to alcohol and high mortalities from all other causes of death;

(iii.) When the influence is infection, due to segregation of workers, as in the case of printers, tailors and shoemakers, the high mortality from phthisis for which the maximum incidence coincides with that for the standard population, is not necessarily associated with a high mortality from any other cause of death.

### Conclusions.

The following pulmonary diseases definitely result from or are predisposed to by dust inhalation: Asthma, bronchitis, pneumonia and phthisis. Asthma and phthisis, considered as pneumonoconioses, are specific diseases, originating from the action of specific dusts. Bronchitis and pneumonia, by way of contrast, result from the mechanical action of dust particles on the air passages, whether the particles possess or do not possess any power to originate asthma or phthisis.

Dust-phthisis is essentially a disease resulting from the chemical and toxic action of silica particles upon the pulmonary tissue. This action gives rise to a pathological state known as silicosis which may terminate fatally; but a fatal termination usually results through the supervention of tuberculosis to which infection silicotic tissue is particularly prone to succumb.

Dust-phthisis is a clinical and pathological entity resulting from the inhalation of silica dust; it can be recognized as such and, therefore, constitutes a condition upon which a claim for industrial compensation may be based.

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## THE PREVENTION OF ACUTE RHEUMATISM IN CHILDHOOD: ITS IMPORTANCE AND DIFFICULTY.

By F. J. POYNTON, M.D., F.R.C.P. (London),
*Physician to University College Hospital, London, and The Hospital for Sick Children, Great Ormond Street.*

ACUTE rheumatism or preferably "rheumatism" in childhood will eventually prove in my opinion to be the keystone of a real advance in our knowledge and treatment of heart disease. I look forward to the time when the attitude of the medical profession toward this problem shows a considerable change in its disposition, to a time when those who are intending to make heart affections their special study, commence their education with a child of under fourteen and not with the adult in whom the scars of disease long past and damage already beyond repair are the adversaries, to a time when the study of details as to the action of digitalis is relinquished for a study of the prevention of the cause of that very condition for which that drug is so often employed, and to the study of the human being with a disordered heart rather than a disordered heart with a human being.

Most of the chief facts about organic heart disease can be learnt in the child and those which cannot, are more easily appreciated from the study of the young heart, as striking departures from the normal. No amount of treatment of heart scars such as we see in the adult heart disease, can do more than patch a damaged pump and important and essential though this is, in the life of a doctor it is not to be compared to the possibilities that surround the prevention of organic heart disease by a deep study of the acute rheumatism in childhood. It is sufficient to read these numbers. I have made notes upon 1,108 cases of first attacks of acute rheumatism in children under twelve years of age and of these 673 had some cardiac affection and 613 chorea.

In England the chief cause of organic heart disease is rheumatism and the incidence reaches its maximum between the seventh and tenth years. The problem of acute rheumatism is then one of great importance and great interest, if of great complexity and difficulty. It is one worthy of the best and broadest minds and one which from its very extent snatches away the blinkers of the specialist and makes him look far and wide for information, experience and help.

### Ætiology.

I need not dwell on the serious nature of acute rheumatism. We are all agreed upon that point and nothing is to be gained by enlarging upon that side of the problem. The causation, however, is a very important question and it is unfortunate that we cannot get further in our knowledge. Dr. Paine and I more than twenty years ago arrived at the conclusion that it was an infection and the infection a diplococcus belonging to the streptococcal group. We pointed out that this was a delicate microorganism not growing readily on ordinary agar-agar or in broth tubes and we refused to call it "specific" because we were very dubious as to whether medical scientists realized what the term specific implied. Our attitude was this: that the disease itself was specific in so far as such a term can be used of human diseases and that the diplococcus was the only bacterial cause we could find of this specific disease and therefore had some special characters.

Now today the position still remains unsatisfactory. Probably the majority believes that the infection is still unknown, but every serious worker at the subject has, I think, been puzzled by the close association of the streptococcal group of microorganisms with active rheumatism.

Why is the infection still thought to be unknown? Chiefly because of the failure of so many workers to find the microorganism in this acute rheumatism of childhood. That in my opinion is the outstanding difficulty and the reasons for this we have detailed in our collected papers: "Researches on Rheumatism." I naturally believe this difficulty will be overcome and the essential cause prove to be streptococcal. From the point of view of histological-pathology the present position of the streptococcal factor is very apparent. If we take a case of so-called subacute bacterial endocarditis, with a previous history of acute rheumatism, cut a section of the damaged mitral valve and compare it with a damaged valve from a fatal acute rheumatic endocarditis, we see in the necrotic tissue of the vegetations a cloud of streptococci, in the latter no such

ance, yet the changes otherwise are in my opinion essentially similar. I hold that in the rheumatic case, if we search sufficiently, we shall often find small groups of micrococci in the tissue bordering on the necrotic zone, but in the simple endocarditis the micrococci perish or are altered in staining properties in the necrotic area. Again Dr. Greenfield, Pathologist to the Queen Square Hospital for Nervous Diseases, investigated by the most recent methods the histology of the brain from a fatal rheumatic chorea. This independent and highly expert work confirmed the changes claimed by Dr. Paine and myself, changes which we demonstrated to be the result of the strepto-diplococcal infection. Thus here again we see that the histological changes in rheumatism are streptococcal in type even in these micrococci cannot be demonstrated.

Much has been written about "Aschoff's nodules," but important though these are, I believe them to be evidence of the well known tendency to healing in rheumatism, not a specific lesion, but a healing streptococcal lesion; upon this point there is independent evidence. None of us can doubt that there is a great, if not effective resistance by man to the rheumatic infection. All clinical evidence favours this view and in my experience there is experimental support also. Lastly if we review the recent literature we find that from almost every rheumatic lesion in childhood one observer or another has isolated streptococci.

The position then seems to me to be this: that if it is going to be admitted that streptococcal lesions are the salient features in children's rheumatism, we have to face these alternatives. Either an unknown infective agent produces a soil permitting the streptococcal infection or the predisposing causes of the disease without the assistance of any unknown infection are sufficient to do this and then the view that the streptococcus is the match which starts the flame, holds good.

At first sight points such as these may appear of purely academic interest, but when faced with a serious problem such as that of acute rheumatism in this country, the uncertain position of the ætiology introduces a sense of insecurity which is very baffling. If the disease is streptococcal and dependent, as I suspect, on predisposing factors, our way is clear, if difficult. A dogged study on a large scale of the streptococci and equally so of the value of predisposing causes, aided by ever improving knowledge of bacteriology, of new methods of research, of biochemistry and not least of clinical facts, must inevitably bring results. On the other hand if the essential factor is some unknown infection which admits the streptococci to the system, we are continually haunted by the feeling that until this is discovered our efforts to combat the disease are being frustrated.

This country with its high incidence of acute rheumatism ought, one feels, to lead the way now in solving the question which has reached such a definite form. There is room for research in many directions. It is a source of regret to me that in the last twenty years no attempt has been made to establish a centre where the great problem of rheumatism would always be in the hands of skilled investigators. A centre from which all over England workers could get in touch with the salient problems, could be warned off a track which has already been explored, and could push forward without losing valuable time by repeating work already thoroughly done.

### Predisposing Causes.

Turning next to the predisposing causes, it seems to me that the time has come to make a closer analysis of them. Discussions on rheumatism are frequent events and on each occasion these causes are very rightly put forward, some with certainty and some tentatively. Now I feel the time has come to try to establish their relative values. Heredity for example, has been claimed and doubted. For myself I have no doubt of its importance which Dr. Cheadle proved from his private case-books. I accept it as a fact and now hope that the biochemist may in the future discover in a case of double inheritance for example some peculiarity in the body chemistry. Damp and cold again, what part do these play? Every student of the subject must form some opinion upon this which may be a right or a wrong one. At present I look upon damp cold as an agent producing the more chronic type of childrens' rheumatism, a type bordering on the rheumatoid. A child in a damp, cold house may not get rheumatism, but if he does I am very doubtful of good recovery while he is living in these surroundings. But do they do more? Does damp cold increase the incidence of the disease? Here it seems to me we want more information, both upon this point and the type of illness that may result.

Next there is chorea. Upon this I hold a definite opinion, but it may be a wrong one. I believe educational strain to be a great factor, but others believe it is the home surroundings to blame or imperfect clothing and footwear which are more important. To this question I attach great importance, because I believe the nervous system in some mysterious way takes a prominent part in this disease. Years ago Dr. Cheadle wrote: "The rheumatic child is the nervous one of the family." Is it because of this nervousness such children are more liable to the infection or is it that they have already a latent infection damaging the nervous system? That rheumatism does damage the nervous system is proven, but we want more evidence, I think, as to whether, for example, in children of rheumatic parentage an unstable nervous system which must have some biochemical equivalent, does not favour the infection.

At the moment my belief is that the present routine official methods of education in vogue are for a type of child a cause of nervous strain, of consequent lowering of vitality and in this way of rheumatic chorea. This incidence of chorea in the school children of our great cities requires in any case careful investigation. I have been told that its occurrence in the board schools is not in nu-

merical proportion more frequent than in the private and public schools of this country, but I am doubtful of the correctness of this statement. We want more facts about this incidence of chorea, for if it is not fostered by school work, then what is wanting in the home management that makes these children fall victims or what other agent is at work? It is the most frequent solitary lesion of the rheumatic infection and this in itself suggests a lack of resistance in the nervous tissues, rather than in any other system and for this feebleness we need an explanation.

Diet as a predisposing cause is another example which illustrates our need for more definite facts. My own view is that diet has probably no vital bearing upon the acute rheumatism of children. Some, however, would cut off meat and others forbid in addition whole milk, egg yolk and artificial sugar, others again attach great importance to eggs and milk as a preventive of rheumatism.

If one authority discards whole milk and another pleads for plenty of milk and both get excellent results, what is the only inference that we can draw? Surely that neither milk nor lack of milk seriously influence the disease. If one authority says give eggs and another says do not give egg yolk and yet both get excellent results and prevent rheumatism, is it not probable that the eggs are not essentially concerned one way or the other? I feed rheumatic children well and early and on the food I would give a healthy child, eggs, meat, fish and so on; my results are not remarkable, but I venture to think on the whole as good as others.

Those children who drift into the rheumatic state and who are continually showing active manifestations over months and years, have not in my practice been children who have had a diet more rheumatic producing, as judged by these authorities, than those who have had a single attack.

It is not the diet that makes a tuberculous case run a relapsing, persistent course, it is something much more subtle and I believe this to be equally true of rheumatism. If, however, I was asked: "Would you not investigate this question of diet and children's rheumatism?" I should say most certainly, though I should not put that particular problem among the first to claim our attention.

I have I think dwelt upon the predisposing causes sufficiently to indicate what I mean by saying that the time has come for us to analyse and weigh the relative importance of all the factors that are claimed to predispose to the disease, rather than to be content with a repetition of our belief or disbelief in them.

The necessary inquiries must take time and will call for helpers of various kinds, family practitioners, school doctors, medical officers of health, health visitors and parents, but there is a useful stimulus to the endeavour in the gradual decline in the severity of cardiac rheumatism after fourteen years. If we can help the young children, we shall very probably be rewarded by their escaping severe rheumatism altogether, and others being spared from the worst forms of carditis.

### Diagnosis.

The clinical difficulty which first attracts my attention if we are considering the problem of acute rheumatism on a large scale, is that of early diagnosis. We may know the chief manifestations of rheumatism, but we also need to recognize how these manifestations usually present themselves.

From the first it seems to me essential to recognize that until the time arrives, if it ever does, that some specific test is discovered, the earliest signs of such a disease must often be equivocal and a matter of individual opinion. We have a sufficient warning of this in the history of tuberculosis. Yet even if we admit that there are and will be difficulties in the early diagnosis of acute rheumatism, I think the present position is such that if we contented ourselves with the greater care of obvious cases, we should still do much good. On this account I am not greatly disturbed by the doubtful cases, believing that with increasing knowledge and interest in the problem the early diagnosis will become more accurate and less difficult. At the British Medical Association meeting at Bath I tabulated the chief onsets that I had met with myself, as follows:

1. The fulminating cases with many manifestations.

2. A large group showing sore throat, muscular pains and arthritis and *morbus cordis.*

3. Another very important group with sore throat, cardiac dilatation and often much general nervousness.

4. Another with chorea and *morbus cordis.*

5. Another with chorea as the predominant and practically the solitary manifestation.

6. A group with heart disease as the primary lesion.

7. Relapsing cases in which one manifestation after another appears and the child drifts into the rheumatic state.

These groups naturally merge into one another, but they help as a general guide. They cover the early history of many of the patients whom we see in London hospitals, but it may well be that in Sheffield or at Bristol or elsewhere in England there are other types that are less frequently met with in the London area or are even peculiar to the district. In any case there remains a number of children who show warning symptoms which disappear or are overlooked, and in this latter case may not come to a doctor until there is very obvious organic heart disease.

This possibility prompted a question I was asked at the British Medical Association meeting. It was this: "If a child has some pain in the knees or hamstring tendons or elsewhere, would you treat this case as one of acute rheumatism and put the child to bed?" You can foresee the sequel. If a confident answer in the affirmative is made, there would be another question of this kind: "Is every ache and pain a child complains about a signal of serious illness?" These questions then bring us at once to the dilemma everyone must find himself in at times in dealing on a large scale with a widespread dis-

ease, until a specific and delicate test for that disease is discovered, a dilemma which can only be dealt with by cautious observation applied to each individual case. Knowing the dangers of rheumatism our attitude is one of watchfulness, but all are aware that not every pain is rheumatic and that every rheumatic pain need not be the signal of a serious illness, but only the sign of a fleeting infection easily overcome by the natural powers of resistance.

The best answer I can give to that question, is that the clinical study of rheumatism in the child should receive the closest attention by students and that the best judges of such equivocal cases are the well educated practical doctors who from an experience not of rheumatism alone, but of disease and human beings in general, can usually detect whether such cases are likely to prove serious or not. This is certain, that in dealing with such a difficult disease on a large scale there must be many errors of observation, but not enough I hold to vitiate a great effort to improve our general management. This too I believe applies not only to the general symptoms, but to the most important one, heart disease. It is not to be supposed that in the absence of a specific test there will be unity of opinion as to whether a heart is organically damaged or not. The early signs of rheumatic heart disease are often so difficult to gauge that there must be differences of opinion. We have only to read Dr. Carey Coomb's valuable book on the subject to recognize this fact clearly enough, but such divergence of opinion does not shake the essential truth that we need to improve our supervision of the child with rheumatic heart disease.

There is another clinical problem which presses, and no study of acute rheumatism could be complete without an allusion to the possible sites of infection. There is little new to be said upon this at the present moment, but at no point in my opinion do we reach the real zone of the difficulty of our subject more obviously. If there is some undiscovered agent, we actually know nothing definite at all about its usual entry into the system. Our evidence upon sites of infection is in part clinical, as for example the well known association of sore throat with an attack, and in part experimental. The strepto-diplococci found in the internal rheumatic lesions are also found in the acute tonsillitis of the rheumatic and will produce in animals carditis, arthritis *et cetera*. These experimental lesions let it be said are not doubtful, they are facts, but many as we know do not accept them as a proof of the essential streptococcal cause of acute rheumatism, yet when you come to other evidence we have none more reliable. Many writers, for example, have dwelt upon the intestinal origin of the disease. Yet if we study acute rheumatism in the child, what convincing evidence have we of such an origin? I admit more rheumatic abdominal lesions than most doctors are inclined to accept and yet if there is one system which has not attracted me as a first cause at fault, it is the intestinal system in childhood. The evidence of a dental causation is for me almost as uncertain. The present position of the local focus appears to be this: that though there is widespread belief that the true cause of acute rheumatism is unknown, many thousands of tonsil and adenoid operations are undertaken to remove the local focus. Either then there must be a tacit admission that it is the strepto-diplococci in the tonsils which are the danger, or there must be an assumption from clinical evidence presumably that the unknown infection lurks in that situation. It is clear then that we need more knowledge upon these points and we can hardly wonder at the diverse opinions expressed by the medical profession upon the propriety of these operations for the prevention of acute rheumatism.

It is not difficult for me to explain my own present attitude, but it may not be the right one. I look upon the tonsils as the most likely structures from their position, to be called upon to deal with the strepto-diplococci which I believe is the actual causal factor. My observations upon the destruction of these micrococci in the tissue cells of man and animals, particularly in the *appendix vermiformans*, lead me to believe that we often escape acute rheumatism because of the protective activity of the tonsils. My observations upon the tonsils also lead me to believe that some are singularly handicapped by their anatomy having large lacunæ which act as culture tubes for micrococci in stagnant exudation. But I am not prepared to say that the tonsils are the only site of infection; in fact I believe that in the naso-pharynx alone there are probably many avenues of invasion.

If, when Dr. Paine and I had finished the work we did on tonsillitis, we had believed that the problem was one identical with that of a small patch of rot in an apple which has only to be cut clean away to prevent further trouble, it is certain that we should have dwelt upon the vital importance of the fact much more insistently. My attitude which logically follows, is this: I look upon the tonsils as useful guardians. Unfortunately as the Great War has shown us, many a brave guardian may win a struggle, but be left so gravely shaken that henceforth he is no longer a guardian but a danger. So with the tonsils; they may prevail, but be left scarred and harbour unhealthy foci and henceforth be a danger and need removal. Removal of healthy tonsils on the other hand because a child has a rheumatic family history, seems to me irrational. Here, of course, will come the primitive question: What is a healthy tonsil? The answer as usual depends on the temperament of the answerer. Some find all tonsils more or less unhealthy, others look upon them as the saviours of society. My answer can only be: Judge them by their behaviour, always remembering firstly that their complete removal does not guarantee freedom even from a severe first attack of rheumatism, secondly that the wholesale removal of tonsils has its own risks. These may be exceptional, but they exist; unexpected death from hæmorrhage, or septicæmia, an acute outburst of rheumatism, grave shock to the child's nervous system and so on. Admitting the rarity of these occur-

rences, I may nevertheless say that I have seen all occur and thus I arrive at my present attitude: a belief in the value of enucleation of the tonsils in chosen cases after careful deliberation and with full knowledge of the present limitations and when I consider them to be failing as guardians and to be a source of danger.

### Treatment.

Coming now to the question of treatment I shall digress somewhat from the main subject to what may be termed a particular incident in the general theme. This is the management of the worst forms of acute carditis in the young. It happens that I have had a wide experience of these cases and I teach students that no condition in childhood requires more discretion and detail in treatment. We are faced by severe general illness in a highly nervous subject. Endocarditis is practically invariable; there are great cardiac dilatation and pericarditis and often enough a certain degree of pericardial effusion. As a result the cardiac dulness extends upwards as well as laterally and may reach a remarkable and alarming size. But this is not all. The left lung is compressed and a massive collapse developes intense tubular breathing appearing first at the level of the lower angle of the scapula. The right lung may more rarely also be involved in this collapse. Even this is not all for with the pericarditis there may be pleurisy and an effusion complicate still more the physical signs in the left axilla and over the left side of the back. From such a condition I have seen remarkable recoveries, but the struggle is a long one and I venture to give you my experience upon certain points in their management. First of all the confidence of the child must be won and the best nursing available obtained. No fussing of the child and no tax on the physical energies short of that which is absolutely necessary are allowable.

The next point is not to do harm with powerful medicines which upset the digestion and cause depression and to gauge the amount of food the child can take without difficulty. If one of these children cannot take food and is sick, the chance of recovery is imperilled. Another guiding line is never to interfere with the pericardium by surgical measures unless driven to it by indisputable evidence. This evidence is very rarely forthcoming in childhood and we must remember that if there have been previous attacks of carditis, the diagnosis between a pericardial effusion and a greatly dilated heart with a thickened, adherent pericardium is almost impossible when the illness is so urgent. In the last case I saw explored for effusion the heart cavities were tapped in three different places.

It has been thought that in the adult early draining of the pericardium would assist recovery and I have seen several cases treated on this assumption. I have not thought, however, that the risk involved was justified by the results and certainly in the present state of our knowledge would not sanction that step in childhood.

It is a great relief to feel that in these grave cases we need very rarely worry about paracentesis of the pericardium. If in doubt about dulness in the left axilla and at the left base, it is wiser to explore the left pleural cavity and much relief may follow the removal of fluid.

If there is any collapse of the left lung we know that with a decrease in the cardiac dilatation and the pericardial effusion that will gradually disappear and we need not fear that sign. It is only a cause of anxiety when it is complicated by pleurisy with effusion or some pneumonic consolidation.

If there are rheumatic pains and arthritis I use small doses of salicylate of soda to relieve the pain, but I do not use this drug if these are absent. My reason is that I have watched and attended patients from the very inception of the intensive treatment based on the specific action of this drug and have arrived at the conclusions that it is not specific and for fragile children is a danger in large doses. Recently I have used "Tolysin," first employed in 1921 by Hanzlik, Scott, Weidenthal and Fetterman. I am not sure that it is a powerful agent, but I have had success in very bad cases in a dosage of 0·3 to 0·42 gramme and have never seen any complications from the drug itself. I have not investigated it thoroughly because of the difficulty of getting supplies, but I believe this drug to be one worth a good trial. The temperature is certainly influenced and more important still the children seem to be generally improved by this drug. I welcome it because with these good signs I have had no anxiety about added difficulties due to toxic effect. At present I have used it almost entirely for patients with the most severe illness and have only lost one; this child died many weeks after the acute attack from myocardial disease.

As a routine I find "Antiphlogistine" comforting and helpful as an application, particularly for delicate children. The after care of such cases we all recognize as demanding the utmost patience and caution.

A question frequently asked is: How long ought a child to remain at absolute rest after severe carditis? One interesting case gives us a hint. A girl who had recovered from a severe carditis, to the extent of surviving the acute illness, had been so damaged that in spite of rest for three months, her heart did not become compensated and she died somewhat suddenly. The necropsy showed a pericardium generally adherent with soft adhesions and a cardiac muscle showing extensive fatty change. This is a proof of the slow recovery of cardiac muscle from severe rheumatic toxæmia. Yet there is no definite time limit, each case must be judged by definite clinical indications, improvement in general health, a steady pulse, a definite impulse, a stable cardiac dulness, an absence of fever and so on. These are the true indications. I am no believer in so-called absolute rest for very long periods for these children. They lose tone and courage and when we are fairly assured of the stability of the heart, we can without danger advance, cautiously testing each stage. If then we happen to be premature, we can at once go back without serious damage.

It is I believe a mistake to keep a child lying absolutely flat, for anyone who tries to lie thus, will find it a greater strain than when slightly raised as in a low deck chair. A child also can then see more easily what is going on around him and have his mind employed. I believe I am right in saying that one rarely sees now the dreadful mistake made in my student days. The acute illness over, the child losing interest, was hurriedly sent home, without special warning or inquiries as to the surroundings. This is disastrous and I think with our present knowledge borders upon malpraxis.

Special convalescent beds in the country have been the object of keen advocacy by me for many years. They will, I believe, do great good and, if they approach the ideal, will prove a valuable step forward in our knowledge and treatment of heart diseases. But there are dangers. Personally I do not advocate, with exceptions that I shall mention, the necessity for cardiac specialists to be in charge of these hospitals. As good a case might be made for neurologists. Neither do we want special hospitals. A section of a general convalescent hospital (not convalescent home) would be equally good. The essential point is that those in charge must be really interested and well trained in the rheumatism of childhood and in the care of children. If not, however good the doctors and nurses, they will not be the best for this purpose. They must have a real human interest in these cases, but seeing that rheumatism attacks all parts of the body a good general doctor is to be preferred to a cardiac specialist.

Nevertheless, in the future it may be possible to have a few ideal hospitals close to great towns, where research can be carried out and all kinds of methods for training the heart and estimating its strength can be carried out.

For these expert cardiologists would be most valuable, both as consultants and as pioneers in fresh methods. In any of these convalescent hospitals or sections no one can doubt that the advice of a cardiologist would be valuable and would, I think, be sought, but to maintain that a well trained, interested general physician cannot look after these children is not my view. If, however, it is thought that the mere bundling of these patients into some country home, to drift along under the fetish of prolonged rest in the country, will be worth while, I frankly state my opinion that it will be a complete failure.

All institutional treatment for children requires the most serious consideration, for even at its best it has certain drawbacks and the experience of rheumatic children in ordinary convalescent homes is conclusive that special care is needed. Suitable education in these convalescent hospitals I look upon as essential, for training the nervous system is a cardinal factor in the treatment of heart disease. Many children can certainly be perfectly well managed at home and I am not advocating in the present state of the country's finances wholesale rheumatic convalescent hospitals.

There is a point about these hospitals upon which I would comment; an idea is creeping in that they are called for on account of the need for open air treatment. I do not consider that to be the real point. The real need is a result of the general non-understanding of rheumatism and rheumatic children. It was thought that these delicate children with this treacherous disease could be managed in ordinary convalescent homes to run about with the usual healthy convalescents.

It is to prevent these disasters that these convalescent hospitals are required and if they can be in the country and if open air can be safely arranged for, all the better. If, however, open air is thought to be as requisite for the rheumatic as the tuberculous, I doubt it and I would take no risks in this climate and would not for one moment make a difficulty over open air as against fresh air.

The next urgent problem in the treatment is the linking up of a stout chain of protection for the future rheumatic children. If we are honest and I hold that in any great step in the advance of medicine honesty is a cardinal necessity, we must admit that we have very little real control over the activity of the rheumatic processes and that there are many children with damaged hearts in this country. Our duty then is clear; it is to give them the best opportunity to become useful and happy citizens. In order to do this the great organizers in our profession, and they abound, must combine to elaborate means by which such children can be helped into suitable employments. A great deal of investigation is required not only to discover these employments and to estimate the good and bad factors in any of them, but to ascertain how to make the suitable ones accessible. Much work is being done, but still more is needed and more effort made to avoid any failure in such organization from friction between various bodies of our profession or from vexing details which are not to the point. A cumbrous certificate for example may lead to overworked hospital officials avoiding them to the detriment of the children affected.

In this part of the problem there will be repeated disappointments met with from the obstinacy or ignorance of the parents, but time and education always conquer in the end.

It must have struck any of us who have been attracted to the subject of this address, how much we should learn of real practical value about heart disease if we could follow the histories of the rheumatic more continuously. For example, why do many of them die of malignant endocarditis? What is it that happens? Is it a change in the infection itself, is there some fault in their usual surroundings, or their occupation or is it again more often another infection which comes as a bolt from the blue? Take again such a commonplace, but vastly important lesion as mitral stenosis. Can we say yet how many of these conditions are arrested with the valve but slightly narrowed or how and why some steadily progress? We get isolated pictures, but we want a great mass of information from life histories traced with some continuity. Here is an ideal illustration. Three years ago I had a girl

under my care in hospital with recurrent chorea and the first clear signs of a commencing mitral stenosis. This child was treated with, I think, all the care possible; she was a long time in hospital and went then to a special convalescent hospital for eight months and later to other country resting places. She is now in hospital again because she was short or breath and I found severe mitral stenosis. There had been no acute illness during those years. In this child the tonsils had been enucleated because of her rheumatism, nevertheless the condition of the mitral valves had made great progress. This is a striking example of the difficult nature of the problem. We appear to have no control over this insidious process and I venture to think we are not clear what is happening in such a case. Is there a ceaseless but most insidious activity of infection which gives rise to no obvious rheumatic signs we yet recognize or are there repeated little outbursts of active disease dependent, for example, upon return to a house which is not really healthy?

There is another point in such cases. Are there certain constitutions particularly in females in which there is a peculiar tendency to contraction in the scar tissue after the active cause for the scarring is destroyed?

I have an idea in my mind, possibly quite erroneous, that some of these cases of mitral stenosis are really rheumatoid affections of the heart comparable to the type of change in some cases of rheumatoid arthritis, where the tissues become withered and stiffened, a condition which may either be constitutional or the result of some peculiar variety of rheumatic infection.

Further difficulties in the treatment are concerned with the climate and locality and more important probably than all, housing. I venture to think that everyone has felt these difficulties. A parent says: "Where can I best take my child to live?" and then your doubts arise. He is on a clay soil, but it is well drained; will the child be better off on a chalky or poorly drained gravel soil: what about the seaside and so on? Again you suspect a house, but families cannot leave houses at a moment's notice and are we encouraged by many modern houses to believe that such a change, if possible, will certainly be for the better?

The medical officers of health may be able to help us, but I can readily understand that their task is a difficult one. If there was partial notification, as has been suggested, this would probably assist them. but I hardly think we are yet in the position to have widespread notification. We need to get a clearer idea of what this problem of rheumatism implies before we embark on such a big step as that.

This concludes my contribution. It is I admit one full of doubts and difficulties, but these are only the natural outcome of dealing with a problem of great national importance and with a disease of great complexity. The salient fact remains as clear as crystal. We must now face the difficulties.

## REMARKS ON CHRONIC GASTRIC ULCER.

By JAMES SHERREN, C.B.E., F.R.C.S.,
*Vice-President of the Royal College of Surgeons, Surgeon to the London Hospital.*

WHEN a chronic gastric ulcer has been definitely diagnosed, there can be no doubt that surgery offers the patient the quickest, surest and most economical method of cure.

It is correct at the present time to say that the majority of these ulcers can be demonstrated by X ray examination after a barium meal. But this is by no means always the case, even when the services of an expert in this type of work can be secured. If therefore the symptoms point to the presence of a chronic ulcer, a negative report after a barium meal should never be allowed to outweigh clinical evidence and lead to postponement of operation. Recently at the London Hospital on one afternoon I operated on three patients in whom definite ulcers were present of the penetrating type in which negative X ray reports had been received. It is no uncommon occurrence to see patients after several courses of medical treatment undertaken after negative X ray investigation and to find on exploration definite chronic ulcers. It is my teaching that these reports are of value only when they confirm the clinical findings and should never be allowed to override them. Like all special methods of investigation it must be looked upon as one link only in the chain of diagnosis.

It may be possible to bring about the healing of an ulcer, demonstrated by X ray examination, by medical means; it is, however, tedious and relapse is usual as the conditions bringing about the original ulcer still exist. It is for this reason that I advise operation in every case of recurrent acute or of chronic gastric ulcer. If we put entirely on one side the certainty of bringing about a cure by operative means and the patient is content with palliation, there is grave risk, if surgery is not resorted to, of serious complications.

Perforation, while not so often met with as in chronic duodenal ulcer, is a very real danger. Hæmorrhage is a greater. While hæmorrhage from a chronic duodenal ulcer rarely leads to an immediate fatal issue, that from a chronic gastric ulcer does so by no means infrequently as a result of erosion of the coronary artery.

The effect of the ulcer on the general condition of the patient is seen in the almost miraculous change brought about in the condition of the patient within a few days of operation, the pallor and worn expression being replaced by a healthy colour and placidity. The wasting brought about by the complication of an hour-glass stomach is a serious feature. Of last but by no means of least importance we come to the development of carcinoma. While this is not so common an occurrence as we at one time suggested, it is supported by abundant clinical and pathological evidence. To take the clinical first. Up to the end of June, 1925, I had operated on three hundred and fifty-seven patients with carcinoma of

the stomach and in one hundred and five obtained a history of attacks of indigestion extending over more than four years of such a type as would have enabled a diagnosis of chronic gastric ulcer to be made on the clinical evidence. I do not meet with this long history as often as I did ten years ago, as at any rate in London chronic ulcers are transferred to the surgeon at a much earlier date and my percentage figures are getting smaller.

There is the pathological evidence. To the end of June, 1925, I had carried out partial gastrectomy for carcinoma of the stomach ninety-eight times; in twenty-four the report received from the Pathological Institute of the London Hospital showed that the carcinoma had started in the edge of a simple chronic ulcer, that is the floor and greater part of the ulcer showed no evidence of malignancy. This, of course, does not exclude the possibility of others having their origin in the same way, the malignant growth having obliterated all trace of the original simple ulcer. These facts appear to me to prove beyond dispute that chronic gastric ulcer is in certain cases a precancerous condition and using the word cause in its widest sense is the only one we know.

The correct surgical treatment of this condition depends upon two factors, the first being the curability of the ulcer by indirect methods, gastro-jejunostomy, the second being the relation of chronic gastric ulcer to carcinoma of the stomach. If it is, as I believe, a precancerous condition, is malignancy liable to supervene in the scar of an ulcer after many years?

There is overwhelming proof that gastro-jejunostomy correctly carried out will bring about the healing of all simple chronic ulcers which are free on the lesser curvature. I have had the opportunity of examining after death from other causes many years later or at second operation for other abdominal diseases twenty-nine patients of my own; in all the ulcer had healed with a thin scar, in some it was discovered only after meticulous search so that had I not personally performed the original operation, I might have doubted if a chronic gastric ulcer had ever been present. But as I have made a practice of always making a personal note in every case wherever the operation was performed, of all details of the ulcer, there is never ground for dispute. In addition I have explored quite as many on whom the operation had been carried out by others, in all the ulcer had healed when an efficient anastomosis had been done. I have on the other hand reoperated on a considerable number of patients in whom the original procedure had been carried out by others and found the ulcer unhealed. These have been cases in which the anastomosis had been made to the pyloric side of the ulcer or opposite to it, or in which the stoma had been made too small or had become so as the result of ulceration, or in which the ulcer had been of the type in which chronic perforation had led to erosion of the pancreas or liver.

In none of the cases I have mentioned in which I found the ulcer healed, has there been any suspicion of malignant diseases developing later.

It is now my practice in the usual small free ulcer on the lesser curvature to carry out a posterior vertical gastro-jejunostomy to its cardiac side in the way I described in my recent Bradshaw Lecture.[1] I have entirely given up any direct treatment of the ulcer in these cases. I have not obtained any better results by combining the operation with excision or Balfour's cautery operation, in fact as both these are liable to result in considerable oozing, adhesions may form and interfere with the free action of the stomach. In all small free ulcers I carry out a simple gastro-jejunostomy; I have never known it fail to bring about healing of the ulcer or known it to be followed by carcinoma at a later date. When, however, the ulcer is adherent, eroding the pancreas or liver, or is for any reason most often that of size, suspicious of carcinoma or if the anastomosis cannot be made to its cardiac side, I carry out partial gastrectomy. This I now do by direct union of the jejunum to the cut surface of the stomach after closing the duodenal end and bring the jejunum in front of the colon with a very short loop and unite to the cut end of the stomach from left to right so that the distal portion of the jejunum is attached to the lesser curvature, the exact opposite to the direction in gastro-jejunostomy. I have found this the most successful partial gastrectomy and the one with the fewest complications. It is simple, rapid and its results are ideal, both immediate and remote. There has from time to time been discussion as to who was the first to carry out this procedure. An improvement of this kind on the Billroth II. operation would sooner or later present itself to the minds of surgeons, but so far as I have been able to ascertain, although there is no doubt it was first published by Pólya, I had performed it several times at the London Hospital before his first case was operated on.

I have recently recorded my experience of the after-history of patients with chronic gastric ulcer treated by simple gastro-jejunostomy[2] and stated that I had never seen the late development of carcinoma in any case of free ulcer on the lesser curvature and that only in three instances[3] did abdominal malignant disease occur after gastro-jejunostomy, the patients coming under observation seven, five and four years after the original procedure. These cases were first operated on in 1910, 1911 and 1912 and were of the type that I now invariably treat by partial gastrectomy. I have not seen the late development of carcinoma in a large number of patients who had their primary operation carried out by other surgeons and who have consulted me for various reasons. I must, therefore, conclude that the development of carcinoma in the scar of an ulcer after it has healed as the result of gastro-jejunostomy is a very rare occurrence and should not influence us in our choice of operation.

There are two complications to which I must allude briefly, they are perforation and hæmorrhage.

Perforation, just as in duodenal ulcer, is now most often met with in chronic cases; perforation of an acute ulcer on the anterior surface of the stomach is a rare condition. Treatment must depend on

the time after perforation at which the patient is seen. The first duty is to save life and closure of the perforation may be all that is possible. It must be remembered that neither in this condition nor in chronic duodenal ulcer is this curative, so that if the necessary curative operation, gastro-jejunostomy or partial gastrectomy, cannot be carried out at the time, it must be done later; it is my custom to reoperate for this purpose in three months.

The immediate treatment of hæmorrhage from a chronic gastric ulcer is morphine, warmth and plenty of fluid, by the rectum at first, later by the mouth. Many of these cases are comparable to secondary hæmorrhage in that after the first effusion there is a latent period in which operation may be carried out; if this is missed, a further fatal loss may occur. I always advise operation as soon as possible after the first hæmorrhage; this is usually in from thirty-six to forty-eight hours, a blood transfusion being given a few hours before, if necessary. In these cases the ulcer should always be directly dealt with, small free ulcers on the lesser curvature by Balfour's cautery method, large adherent ones by partial gastrectomy. In this way we can be certain that we have dealt with the source of the bleeding.

In conclusion I would state that, carried out on these lines, the surgery of simple gastric ulcer gives, I think without exception, at least as satisfactory results as the surgery carried out for the relief of any abdominal condition.

#### References.

(1) James Sherren: "Bradshaw Lecture on Gastro-Jejunostomy," *The Lancet*, November 14, 1925, page 1007.
(2) James Sherren: *Ibidem.*
(3) James Sherren: "Hunterian Lecture on the Late Results of the Surgical Treatment of Chronic Ulcers of the Stomach and Duodenum," *The Lancet*, March 27, 1920, page 691.

## WASTING AS A SYMPTOM: A CLINICAL LECTURE.[1]

By ROBERT HUTCHISON, M.D., F.R.C.P.,
*Physician to the London Hospital.*

I PROPOSE in this lecture to discuss the significance of wasting or loss of weight as a symptom in disease. Now all organic diseases tend, of course, to be accompanied by wasting, but I am now speaking of wasting when it occurs as the "presenting symptom," that for which the patient chiefly consults you. And by "wasting" I mean essentially a progressive loss of weight. You will be consulted often enough by patients who think they are "too thin" or whose friends or parents think so for them, but these are not cases of wasting. Such persons have always been too thin and are often in spite of this —or perhaps because of it—in very good health. If there has been no recent decline in weight such cases may be left alone. For it is important to understand that there is no constant standard of weight for any given height notwithstanding the tables of Hutchinson and others which seek to establish such. The sole criterion is what each individual knows his own weight to be when in health; if he has not fallen below this there is no need to worry.

In order to appreciate the significance of loss of weight we must understand what it is possible to lose. The three "variables" in the body are (i.) water, (ii.) fat, (iii.) muscle and loss of weight may be due to loss of any one or all of these.

The importance of water as a factor in weight is often forgotten in clinical medicine, although at least two-thirds of the body-weight consists of it and in experimental starvation it is found that 66% of the loss consists of fluid. It is probable that individual differences in weight even in health are due to a larger degree than is supposed to differences in "wateriness." In old age there is a tendency for the tissues to shrink and dry up and prolonged residence in the tropics often has a similar effect. Large drinkers, on the other hand, especially beer drinkers, tend to be "juicy" and a relatively large proportion of their weight is made up of fluid.

1. Very rapid loss of weight due mainly to loss of water—a dehydration of the tissues takes place. There may be several causes of such dehydration:

(*a*) Diarrhœa, especially if the stools are very fluid, is a cause. This is well seen in summer diarrhœa in infants and to a still more pronounced degree in cases of cholera.

(*b*) Vomiting is another cause, especially where large quantities of fluid are brought up. Most of the loss of weight in cases of pyloric stenosis, for instance, is due to dehydration and the same process accounts for a rapid fall during the occurrence of a gastric crisis in tabes.

(*c*) Sweating, if profuse, may lead to a considerable loss. As is well known jockeys have recourse to this method (sometimes combined with purging) in order to reduce weight before a race and fat people who lose a few pounds after a Turkish bath often deceive themselves with thinking that they are "thinner" in consequence when really they are only "drier." The decline in weight which some people experience during summer weather or after going to live in a hot climate, is in part at least due to a similar process of desiccation and in any case of disease attended by profuse sweats a loss of weight from this cause may be looked for.

(*d*) Profuse diuresis is another cause of dehydration and any polyuria, no matter how brought about, leads to a certain amount of wasting. In the following case, for examples, the polyuria of chronic interstitial nephritis had led to a rapid loss of weight which was the main reason for the patient seeking advice.

CASE I.—A man of forty-six complained of rapid loss of weight during the past few months; he also felt weak and depressed. He attributed his symptoms to an attack of "ptomaine poisoning" a year before. On examination he was thin but looked healthy. His systolic blood pressure, however, was 250 millimetres and he had an abundant low gravity urine containing a trace of albumin.

I remember another case in which an abrupt fall in weight combined with profuse polyuria and thirst

[1] A lecture delivered at the London Hospital.

occurring in an elderly man with "prostatic kidneys" led to an erroneous diagnosis of diabetes.

Just as a sudden loss of fluid from the body may be mistaken for a loss of fat or flesh, so the rise in weight which results from retention of fluid, may be erroneously ascribed to a gain of fat or flesh. Such a rise occurs in some cases of cardiac disease (for example the alcoholic heart) and nephritis owing to the presence of "latent" dropsy—a general increase in the fluid content of the body without demonstrable œdema. As the result of treatment this fluid may be rapidly lost with a consequence fall in weight. The gradual development of ascites in a case (say) of carcinoma of the stomach or of tuberculosis may similarly cause the weight to rise and this may deceive the doctor into thinking that an improvement in the patient's condition has set in. An increase in the osmotic tension of the tissues is another cause of retention of fluid in the body with a rise of weight. This increase may be the result of an excess of salt in the diet or of the administration of drugs, such as the iodides or bicarbonate of soda. For instance, I have known patients suffering from diabetes with ketosis to whom large quantities of bicarbonate of soda were being administered, to gain weight rapidly and thus to be attributed to an improvement in the general nutrition until the development of actual dropsy made the true cause of the gain evident. When the drug is stopped such patients rapidly lose weight again.

2. Loss of weight may also and more commonly be due to a diminution of the other two variables, fat or muscular tissue in the body. It is impossible clinically to distinguish between these two forms of loss, but if great weakness accompanies the decline in weight, some loss in muscle may be inferred and this is always more serious than a mere loss of fat. For practical purposes, however, the two forms of loss may be considered together and we have now to discuss the possible causes of wasting due to one or both of them. The cause may be either (i.) organic or (ii.) functional.

Of organic causes the chief are (*a*) early tuberculosis, (*b*) hyperthyreoidism, (*c*) glycosuria, (*d*) carcinoma.

(*a*) Early tuberculosis—pulmonary usually—is to be thought of especially in young adults and the other symptoms and signs of the disease should be carefully inquired into.

(*b*) Hyperthyreoidism is, I believe, a very common cause of rapid wasting and one which is often overlooked. It is most frequently met with in young women and middle-aged men. The other symptoms and signs to look for are general "nervousness," a fullness or enlargement of the thyreoid and slight prominence of the eyes (though both these signs may be absent), tachycardia and fine muscular tremors, the two last signs being very constant. Here is a case in point:

CASE II.—A woman of thirty-seven felt "seedy" and had lost a stone in weight in the past month. Her previous health had always been good and she knew of no cause for her condition. On examination she was still very well nourished, but there was slight exophthalmos, her pulse rate was 104 and she had a fine tremor. The thyreoid could not be felt and she seemed otherwise sound. The diagnosis was hyperthyreoidism.

(*c*) Glycosuria as a cause of wasting is chiefly found in elderly persons; in the young diabetic loss of weight is not usually the presenting symptom. The following is an illustrative case:

CASE III.—A man of sixty-five came complaining that he had been rapidly losing weight for the past three or four weeks. He had no other symptom and his previous health had been good. He was still fairly stout, but his skin was loose and he had evidently wasted. His weight was 66·5 kilograms (ten stone five pounds). Physical examination revealed nothing except that the urine (specific gravity 1025) contained a small amount of sugar. Under a restricted diet the sugar rapidly disappeared and he gained 680 grammes (one and a half pounds) in weight in ten days.

(*d*) Latent carcinoma is sometimes a cause of wasting as the chief symptom especially, of course, in old people. The stomach is the organ most likely to be involved and weakness, loss of appetite and dyspepsia are the other symptoms to be inquired for. An X ray examination and a test meal may furnish confirmation.

Of the functional causes of wasting the chief are:

(*a*) Underfeeding. Not uncommonly loss of weight is simply due to this cause. I saw a great many such cases towards the end of the war in patients who had "rationed" too conscientiously and with the abolition of rationing, the loss was usually quickly made good. Sometimes in dyspepsia patients the wasting is due to overdieting either "on their own" or from too strict an obedience to the advice of their doctors. In other cases it is the result of some obsession which leads to voluntary starvation. In women this often takes the form of a fear of getting stout and I have seen several cases of severe wasting result from this. In one case, that of an intelligent but morally priggish young man, the refusal of food was due to the belief that eating increased the tendency to nocturnal emissions. He became so much emaciated in consequence of his abstinence that he nearly lost his life.

(*b*) Frequently the cause of wasting can only be spoken of as nervous—the result of mental and emotional strain. Thus we speak of a person being "worn with anxiety" or of grief as "preying upon" one. "Affairs of the heart" are specially apt to have this effect and it seems to have been common for Victorian young ladies when disappointed in love to go into a "decline," though this seems to be a less frequent occurrence in our own days. The following were cases in which the loss of weight could only be ascribed to emotional causes:

CASE IV.—A woman of twenty-seven complained that she felt tired and that her weight had gone down nineteen kilograms (three stone) in six years. She was a migraine subject, but had otherwise had good health. She was the third wife of an elderly man, childless and much left alone; she "disliked the place she lived in" and worried about it. On examination she was a thin, tired-looking woman with some visceroptosis, but otherwise sound. She had had "rest cures" at home without benefit, but on going to stay with friends at the seaside she gained twelve and a half kilograms in weight in the course of a few weeks.

CASE V.—A girl of sixteen had been "getting thin" for six months. Her appetite and digestion were good, but there had been amenorrhœa for more than a year. Her mother thought that "school worried her." On examination

her weight (clothed) was only thirty-four kilograms (five stone five pounds); she looked emaciated but not cachectic, her skin was dry and harsh, but all the organs were healthy and the urine normal.

She was ordered a Weir Mitchell course and under this gained six and a third kilograms (a stone) in a month and ultimately got quite fat and well.

There are probably several ways in which such perturbations of the nervous system may cause wasting. They usually tend to impair appetite, but they may also, perhaps, increase the activity of the thyreoid and they may even interfere directly with nutrition through the lessening of a trophic influence.

(*c*) The most severe wasting of a functional sort is that met with an *anorexia nervosa*. This condition, as is well known, occurs only in hysterical young women and often owes its origin to an emotional shock. The chief symptoms are great and progressive wasting, accompanied by any proportional loss of strength, an obstinate refusal of food and disappearance of the menstrual flow. It is often difficult to discover why these patients refuse food. The trouble may start, as indicated above, after an emotional shock, such as the death of someone to whom the patient was much attached, or it may have come on after an unhappy love affair. Sometimes it arises out of voluntary starvation from fear of getting fat. In one of my cases the patient was so disgusted by the development of the breasts at puberty that she thought she would starve them away! In every case there is something at the back of the patient's mind if one can only get at it.

Patients with *anorexia nervosa* get extremely thin, but seeing how little some of them eat or appear to eat, it is often a puzzle how they remain alive at all. Some of them, no doubt, are like the young lady called Maud of whom it was said, you remember, "that to eat much at table she never was able, but when in the kitchen, oh Lord!" But although surreptitious feeding may account for the survival of some, yet in many this can be ruled out as an explanation and one can only suppose that the patient developes a very low basal metabolism and "runs the machine" with the maximum of economy. The wasting of *anorexia nervosa* is the only form of functional loss of weight which may become dangerous. I have known several cases of it in which the patient developed an acute pulmonary tuberculosis.

(*d*) Some cases of wasting in the absence of organic disease seem to be toxic in origin. The excessive use of tobacco certainly causes loss of weight in some persons. Calverley reminds us:

> How those who use fusees
> All grow by slow degrees
> Brainless as chimpanzees,
> Meagre as lizards,

and I recall the case of a doctor who gained 2·7 kilograms (six pounds) in weight in a few weeks after giving up cigarettes. Even more potent in this connexion is opium; nearly all morphine addicts are wasted. I remember the case of another medical man whom I used often to meet in consultation many years ago. Every time I met him he seemed to be thinner than before, though otherwise he appeared healthy enough. Ultimately, when I was beginning to be seriously alarmed about him, it "transpired" (as the newspapers say) that he was addicted to the use of morphine which, of course, explained everything.

I may conclude with a word or two about the treatment of wasting. In organic cases, of course, the treatment is that of the causal disease, but in functional cases the symptom has to be dealt with directly. The mistake commonly made is not insisting upon the patient going to bed. It is quite impossible to get many of them to put on weight as long as they are going about; this is particularly true in all patients with visceroptosis. Further, treatment is much more likely to be successful if carried out in a home or hospital than in the patient's own house. In cases of *anorexia nervosa*, indeed, not only must the patient be removed from home, but she must be isolated from all her relatives during the treatment on strict Weir-Mitchell lines. In the other cases, however, in which there is no hysterical element, complete isolation is not necessary, though the number of visitors may have to be restricted.

However been got to bed the patient must be gradually fed up. If emaciation is extreme, it is advisable to go cautiously with the feeding at first as the digestive power in such circumstances is often greatly impaired. By degrees, however, food is pushed until a full diet is being taken.

The fattening cure may take from four to eight weeks or longer and may be combined with massage and other physical treatment as well as any form of psychotherapy which seems indicated. A good deal of the weight gained, especially at first, consists of water and this portion will be largely lost again when the patient begins to get about, but there should also be a substantial and permanent gain of fat and flesh. There is no need, however, to aim at going beyond the patient's "normal" weight.

In some cases the effect of the fattening cure is altogether transient and the only way of permanently improving the patient's nutrition is by a complete change in his mode of life. Here is an example:

CASE VI.—A man of thirty-seven complained that he had lost 6·35 kilograms (a stone) in weight in the preceding six months; there was also some fullness and discomfort after meals, but no other symptoms. Physical examination was entirely negative. He was employed as a clerk in a city office and did not think that the life suited him. After the failure of one or two fattening cures, he took up more active work in the country and in six months had gained 12·7 kilograms (two stone).

## CHANGING CONCEPTS CONCERNING ORAL SEPSIS.[1]

By EDWARD C. ROSENOW, M.D.,
*Division of Experimental Bacteriology, The Mayo Foundation, Rochester, Minnesota, United States of America.*

IT seems to me appropriate to consider on this occasion the development of this important subject, the clinical and experimental observations on which

[1] Address delivered on the occasion of the award of the Callahan Memorial Medal by the Ohio State Dental Society at Columbus, Ohio, December 2, 1925.

it rests, and the good that has already come from its application, and to estimate the benefit that may confidently be expected to accrue from it in the years to come. Moreover, since your commission has seen fit to present this award to me for efforts of my own in this field which bear so directly on dental problems, I may be pardoned for reviewing briefly results of my own studies, particularly the earlier experiments which led to the conception of the theory of elective localization, and to point out why investigators, aside from those who have worked with me, until recently have had difficulty in corroborating the experimental findings.

Nowhere in medical history is the value which comes from combined clinical observation and experimental inquiry better illustrated than in the development of our knowledge of focal infection. Notwithstanding the repeated suggestions made in previous years concerning the ætiologic relationship, especially between acute localized infection and grave systemic disease, the medical and dental professions as a whole remained indifferent until Billings and his coworkers made their clinical observations and correlated experimental studies in animals demonstrating the importance of localized infections, even though small and symptomless, as common sources of various systemic disorders. The broader conception of this interrelationship, well expressed by the term "focal infection," may therefore be regarded as having originated in recent years. I count myself fortunate in having been associated with Dr. Billings and to have been able to participate in this earlier work in Chicago and to have had the opportunity to continue my investigations in conjunction with clinicians at the Mayo Clinic in Rochester.

Aside from affording information concerning the pathogenesis of a series of diseases formerly not well understood, the work gave medical science a therapeutic principle of the utmost importance. Numerous reports on the relief of symptoms following the removal of foci of infection soon followed. The scope of this influence may be judged by the voluminous literature on focal infection which has since appeared throughout almost the whole civilized world. The benefits which have already accrued directly or indirectly, are almost beyond calculation. It is not surprising that much speculation and opinion, often insufficiently supported by facts, have been indulged in, both by clinicians who often have little conception of the real significance of experimental studies and by purely laboratory workers who know so little of the clinical applications of their findings. All of the men who were associated with Billings and who did the experimental work had had both clinical and laboratory training and had intimate knowledge of the condition of the patient under investigation. Both types of study have value, but this could be greatly increased if the studies in this field could be made in collaboration or in closer cooperation. Such combined study is the crying need today in the further working out of the problem. How much more might be accomplished if the many operators in the dental field, particularly root-canal experts, exodontists and dental surgeons and specialists generally, could correlate their clinical and Röntgenological findings with results of equally expert bacteriological and experimental studies in animals than by attempting to evaluate the safety of the various procedures, such as root-canal work, by mere clinical observation, as is now almost universally done. It is a noteworthy fact that the results of my work in this field have been verified almost wholly by men who have had the necessary clinical guidance in the selection of material from patients, or who have had extensive clinical experience as well as a laboratory training.

The inability of certain investigators to corroborate my results would seem to be explainable on the ground of the improper selection of cases and material for study or of insufficient attention to technical details, as pointed out especially by Gay and Haden. The early results in studies on ulcer were corroborated by Hardt and Helmholtz, those on iritis by Irons, Brown and Hadler and those on cholecystitis by Brown. The elective localizing power of streptococci and colon bacilli from urinary infections has been demonstrated by Helmholz and Beeler. The results in the study of ulcer and arthritis have been fully corroborated, extended and controlled in my laboratory by Meisser, with streptococci isolated from infected teeth in the study of ulcer of the stomach and arthritis, by Nakamura with streptococci isolated from extirpated tonsils after death of the patient. My methods have been used successfully in a study of the ætiology of pyelonephritis and ulcerative cystitis by Meisser and Bumpus, by Moench in a study of the ætiology of endocervicitis, in which she demonstrated that this condition is often due to partial-tension streptococci and may be a common cause of arthritis in women, and in a study of the ætiology of chronic ulcerative colitis by Bargen. Price working independently has also reported corroborative results on ulcer of the stomach and on pyelonephritis, as concerns dental foci especially. The work on iritis and other diseases of the eye, has been verified and greatly extended independently by Haden. Through strict attention to technical details he has even succeeded in producing onychia in rabbits with streptococci from foci of infection in patients suffering from multiple onychia, an example of extreme specificity.

Certain investigators contend that all infections are focal in character. It is true that this is often the case in the microscopic sense, yet there is a fundamental difference between an area of infection around a joint, as in chronic arthritis for example, and infection in the jaw surrounding devitalized or diseased teeth or in the tonsils containing dilated crypts with a narrowed or plugged orifice or an encapsulated abscess, which for mechanical reasons cannot heal or drain.

In the systemic or secondary focus the number of organisms is usually small, whereas in the primary foci mentioned the number is usually large. Again, there are those who say: "Why care about infected pulpless teeth or other foci of infection since the

mucous membrane of the upper respiratory tract and especially of the intestinal tract harbours millions of organisms?" It is well known that normal mucous membranes are relatively impermeable to microorganisms. In order to make the analogy more nearly correct, I would venture to suggest that the nerve supply to various segments of the intestine be cut off and that certain parts be wholly or partially ligated. Indeed, abundant evidence is already at hand in which systemic disease results from improper functioning of the intestinal tract from various causes, such as reversed peristalsis, kinks and adhesive bands, infected diverticula, and especially from an appendix whose orifice or lumen has become narrowed from scar tissue, the result of localized infection or has become plugged by focal concretions.

Clinical observations indicate that localized infections in certain structures, such as tonsils, teeth and sinuses, are more likely to be associated with systemic effects than those in other structures, such as the lung in bronchiectasis or in the urinary tract in cystitis and pyelitis. This may be due to a difference in the kind, number or invasive power of the bacteria in these locations or to peculiarities of the tissues harbouring such foci. In general, it may be said that the harm which is prone to come from foci of infection, is directly proportional to the lack of drainage to the surface. The more virulent the bacteria, the less they need a gross focus for entrance and the lower their virulence, the greater is the factor of focal infection in the production and maintenance of chronic disease. However, there is much clinical evidence indicating that foci of infection, as in tonsils, are often directly responsible for making attacks of acute infectious diseases more severe than they would otherwise be and for increasing the incidence of complications, such as in diphtheria and scarlet fever. Foci of infection in the upper respiratory tract, as in the tonsils, may also be of epidemiologic importance, as is indicated by the suddenness with which the diphtheria carrier state disappears after tonsillectomy. In fact, the question of focal infection in its broader sense is as wide in its scope and as difficult of proper application as is the practice of dentistry, medicine and surgery combined. It cannot be applied by rule of thumb any more than can the healing art. A focus of infection that for mechanical or any other reason cannot heal or drain, that is teeming with organisms often in mixed culture, must ever be considered not only as a favourable place of entrance but also as a good place for bacteria to maintain or acquire high and particular invasive powers. Clinical observations support these contentions and experimental results have gone far to establish them. Thus, control experiments with cultures from the buccal mucous membrane, the surface of the tonsils and throat often proved "negative," the animals usually remaining well and entirely or relatively free from lesions, whereas strains from the depth of the focus produced characteristic lesions. Moreover, direct experimental proof of this fact has been obtained in the production of chronic foci by the devitalization and infection of teeth in dogs. Strains from patients with arthritis, nephrolithiasis and ulcer of the stomach, having elective localizing power, often retained this property for many weeks and months in the periapical structures of the teeth so infected, whereas several aerobic platings sufficed to destroy it completely.

### Theory of Elective Localization.

The best proof of the ætiologic relationship of a focus of infection to a given lesion is the production of the lesion in animals with bacteria isolated from the focus in the patient.

Through the use of special cultural methods in which due consideration was given the question of oxygen tension and the injection of animals with the freshly isolated strains, extremely characteristic localizations were obtained with *Streptococcus viridans* from patients with subacute bacterial endocarditis and with streptococci isolated from the joints of patients with rheumatic fever before the idea of elective localization occurred to me. The peculiar localizations obtained were considered as being due to different species of streptococci, rather than to peculiar temporary properties of different strains of the same species. It was not until ulcer of the stomach was produced in animals during my study on the transmutation of pneumococci and streptococci with "laboratory" strains that had attained a certain grade of virulence from successive passage through animals, that the theory of elective localization took definite form. The long series of experiments in animals that have been done since by myself, my pupils and independent workers, leaves no doubt that the elective localizing power of the bacteria in foci of infection largely determines the location of the systemic lesion or disease a person with foci of infection is likely to develope. This statement should not be taken to mean that this always occurs independently of predisposing factors, however. Exposure, trauma and fatigue of certain structures, improper food and bad sanitation, lack of sunshine, alcoholism and other excesses and heredity undoubtedly lower the threshhold of local or general resistance and thus greatly increase the likelihood of elective localization of bacteria and other infective processes. Direct intravenous injection of bacteria which had grown in foci, such as those contained in small amounts of pus from tonsils, has often been followed by localization with the production of lesions corresponding to those in the patient. Moreover, the specific affinity was sometimes so marked that it was not always necessary to give the injections intravenously; this observation answers an objection raised regarding large numbers of bacteria injected in routine work. Introduction of the bacteria in suitable dosage into the peritoneal cavity, the trachea, the brain, the stomach, the rectum or into the nasal cavity by packing the nose with gauze soaked in the culture, was followed by specific localization in certain instances, especially when the more virulent strains were used, thus supporting the clinical observation that systemic disease is not always attributable to a demonstrable focus of infection.

In order to remove all doubt regarding the importance of latent foci of infection as an important factor in the production of disease, Meisser and I produced latent foci by devitalizing and infecting the teeth in dogs, thus closely simulating the conditions often inadvertently induced in persons by dentists. Nephritis, nephrolithiasis, ulcer of the stomach, spasms of the diaphragm and other muscles and chorea have been produced in this way with cultures isolated from patients with these respective diseases and in each instance the causal relationship of the organism introduced into the teeth to the metastatic lesion, has been established by the demonstration of the organism in the lesions and focus and by the elective localizing power of the strains isolated at the end of the experimental period. During these latter experiments another important fact was noted. The bacteria in the induced latent focus of infection besides producing the characteristic disease, appeared to exert general deleterious effects. The animals lost weight and became more susceptible to intercurrent infections, although they were kept under conditions identical with those of control animals. In other words, conditions of hygiene and diet that were inadequate to maintain weight and health in normal dogs, were inadequate for dogs with latent foci of infection. The harm, therefore, from improper food or sanitation may be greatly exaggerated by bacteria harboured in foci of infection, a point not yet sufficiently considered.

In my experiments still other important facts have been brought to light. Streptococci having the same specific localizing power were demonstrated in more than one focus in the same person simultaneously and in some instances at intervals in one or more foci over a long period. These experimental results are in harmony with the common clinical observation that persons affected with a particular disease, such as iritis, ulcer of the stomach or arthritis, are prone to have recurring attacks of the same disease which often tends to become chronic in character. In the light of these facts the possibility that the tissues or fluids of the body afford the conditions on which the development of the peculiar localizing power of bacteria depends, must be seriously considered. The hereditary tendency observed in some of these diseases also points in this direction, as does the repeatedly-made observation that a higher incidence and more marked lesions occur in the characteristic organ or tissues of animals infected with the organisms from foci at the time of acute attacks or during acute exacerbations in chronic affections than during quiescent intervals. Moreover, well marked differences in the incidence and severity of specific lesions were noted at different seasons of the year, especially in chronic ulcer and arthritis. During the colder months when exacerbations and an increased incidence of these diseases are so common, the incidence of positive results in animals was appreciably higher than during the warmer months, when patients were relatively or entirely free from symptoms.

The fact that bacteria of the same species localize electively, depending on the degree of virulence or other acquired property, is no more remarkable than the fact that bacteria of different species tend to localize in particular organs or tissues. The loss of virulence of streptococci or other bacteria on artificial cultivation and its increase on passage through animals, are well recognized. The change in localizing power likely occurs for the same reason.

The reasons for the elective localization of bacteria are still obscure. No doubt the same principles apply that determine the localization or pharmacologic action of chemicals and drugs. Indeed, my experiments in ulcer of the stomach and epidemic hiccough strongly support this hypothesis. It was found that strains of the streptococci from patients with ulcer which had elective affinity for the mucous membrane of the stomach and which produced ulcer, on intravenous infections elaborated a poison or toxin within the bacterial cell and in the broth which had specific damaging effects. Injection of the washed dead bacteria and filtrates of actively growing cultures produced hæmorrhage and ulcer of the stomach without inciting lesions elsewhere. Even more striking were the results following intracerebral infection of living cultures of the streptococcus from patients with epidemic hiccough, the dead bacteria and filtrates of freshly isolated cultures. In each instance spasms of the diaphragm or other muscles were produced. Moreover, filtrates of nasopharyngeal washings and pus from tonsils at the time of attacks sufficed to provoke spasms, whereas similarly prepared filtrates after recovery were without effect. Aside, therefore, from the specific attraction or tropism of certain cells or tissues for certain bacteria, their localization and growth in certain tissues are dependent on the production of toxins or poisons which damage those tissues specifically. The specificity of some of the strains was so marked that intravenous injection in pregnant rabbits was followed by localization and lesions similar in fœtuses and mother rabbits. The view held by obstetricians that foci of infection predispose to miscarriage or to the ill-health of the fœtus is supported by clinical and experimental findings. The far-reaching deleterious effects which focal infection may have even in conditions generally thought to be hereditary in origin, are indicated especially by the work of Talbot who says:

> The evidence tends to show that most congenital malformations which are the results of lack of embryonic development are not hereditary defects but acquired *in utero;* that defects in development are due to injury to the placenta during the early weeks of pregnancy; that the injury to the placenta is due to maternal hæmatogenous infection of the blood vessels of the placental site, and that the source of the hæmatogenous infection is generally to be found in the teeth and tonsils.

### Practical Consideration.

The practical applications of the principles of focal infection and elective localization are fraught with many difficulties. Systemic diseases, once thoroughly established and often associated with anatomic changes that in themselves may continue to give rise to symptoms even though there are no organisms present, may continue after all evident foci are removed. The instances of cure or arrest

of progress in systemic diseases by the removal of foci of infection are so numerous that search for and removal of all foci possible is indicated in almost every case of serious systemic disease for which there is good clinical or experimental evidence of focal origin. In a given case the variations in the invasive power of the bacteria, the wide differences in natural or acquired resistance to microbic invasion in different persons and at different times, the age and sex, the history of previous attacks, the duration and character of the disease from which relief is sought and hereditary tendencies must all be considered in determining when and whether all or certain foci should be removed or whether the condition is indeed of focal origin.

To arrive at a correct diagnosis and to manage focal infection properly often require the advice of specialists in the various branches of the healing art, as well as that of a competent bacteriologist. The opportunities of the dental profession have been greatly increased through the better understanding of the problem of focal infection in general and especially of the dental area. Many problems are still unsolved, but enough have been solved to prove that prevention and elimination of oral sepsis should henceforth take precedence over preservation of the teeth which has been practised in the past, almost wholly for mechanical or cosmetic purposes. Preventive measures should begin in childhood with a view to obtaining proper development of the teeth and oral cavity. The principles underlying various procedures for the prevention and cure of infections of the gums and enveloping membranes of the roots of teeth are fairly well understood and effectively applied by many.

Infections of the dental pulp, pulpless teeth and apical abscesses are theoretically the most dangerous of the various forms of dental foci. They are usually free from symptoms and hence unsuspected. They are situated in osseous tissues which allow no expansion. They can drain only into the circulation and are exposed to pressure transmitted by the teeth during mastication. The infection remains active for years and the bacteria are not encapsulated as is usually assumed, but are found in areas of active inflammatory reaction where the formation of new blood vessels affords drainage into the circulation or lymph stream.

The bacteriologic study and the animal experiments carried out by myself, by Meisser and others working with me, by Price and especially by Haden, prove that vital teeth free from caries are usually sterile, but that practically all pulpless teeth removed from patients are infected and that with the organism isolated, usually streptococci, the disease causing the patient suffering may often be reproduced in animals. The infection in pulpless teeth is usually present whether or not the teeth show rarefaction in the Röntgenogram. Moreover, it does not seem to make much difference whether the root canal has been "properly" filled or not. The idea of focal infection has often been wrongfully condemned because one or more pulpless teeth that showed no rarefaction were thought to be sterile and left untouched or when foci elsewhere were not even considered. Again, simple extraction of infected teeth, while usually sufficient is not always so to eliminate infection in the jaw. This seems peculiarly true if teeth have been rendered pulpless artificially. In the individual case that method of removing infected teeth should be chosen which assures elimination of the infection which is least destructive, and which is associated with the lowest incidence of constitutional reactions manifested by fever and exacerbation of the systemic condition. Exacerbation following extraction of teeth should be regarded as experimental proof that the infection around the teeth removed has causal relationship and that in all likelihood not all of the infected tissue was eradicated during the operation.

A tooth from which the pulp has been removed or which has died from infections, seems to become a place of lowered resistance to bacterial invasion. The methods generally used in root-canal work are certainly not adequate to prevent subsequent infection. The wholesale devitalization of teeth and the filling of root canals often for trivial reasons as practised in the past, considered in the light of our present knowledge, should be regarded as veritable experiments on human beings. It is greatly to be hoped that an efficient method may be found that will not only sterilize pulpless teeth and periapical tissues that have become infected, but prevent subsequent infection, especially of the periapical tissues. The fulfilment of this latter requirement seems almost unattainable. Until this has been accomplished it would seem wiser to remove teeth that have become infected or that require extirpation of the pulp, than to retain them and have them become a source of infection later. No one deplores more than I the ruthless extraction of teeth which has been practised in some instances as a result of the work on focal infection. Vital teeth free from pyorrhœa should never be extracted, except as it becomes necessary for restorative work. The extraction of pulpless teeth seems to me to be indicated, even regardless of the appearance of the Röntgenograms, in cases of serious systemic disease for which no other focus can be found. Good effects are especially prone to follow in cases in which it is possible to reproduce the disease in animals with the bacteria isolated. The results from the use of vaccines prepared from the strain proved to be guilty are also often strikingly favourable. Since the diseases which are commonly the result of focal infection, are usually due to streptococci, immunity to which is of short duration, not too much should be expected from the use of specific vaccines or serums. They cannot take the place of removal of a cause, the focus.

The removal of foci of infection, particularly if the focus is situated in tissues which may be readily spared, such as tonsils and teeth, should be practised in certain instances not only as a curative but as a preventive measure. General deleterious effects, including lowered resistance to intercurrent infection, as well as specific localization, occurred in dogs in which focal infection had been induced artificially in teeth. This may be regarded as fur-

nishing an experimental basis for the improvement in general health so commonly observed and for the favourable results obtained in the treatment of diseases such as diabetes, syphilis and pulmonary tuberculosis following the elimination of foci of infection.

A careful consideration of all the facts now available indicates that a sane and comprehensive effort toward the prevention of septic foci and their cure wherever found, will often result in the prevention and cure of chronic disease, in the alleviation of human suffering, in a better preservation of the tissues in old age, in a longer average duration of life, in increased mental and physical efficiency and through the laws of heredity make for a sturdier race. Since focal infection is so common in the teeth and surrounding structures, the dental profession may confidently be expected to do its full share in the preventive and curative medicine of today and of tomorrow.

# Reviews.

## FUNCTIONAL NERVOUS DISEASE.

AFTER the war Dr. Bousfield held the position of lecturer to members of the Pension Boards, as it was realized that nervous disorders were extremely important, yet little understood by the general practitioner. He has amplified and rearranged his lectures and published them under the somewhat ambitious title for so small a work of: "The Pathology, Diagnosis and Treatment of Functional Nervous Diseases."[1]

The author commences by explaining the foundation of consciousness on tension: "Pain represents the conscious manifestation of a state of a tension and pleasure, the conscious manifestation of the reduction of tension." His theory is ingenious and interesting; though it is far from complete it will probably be found of use in explaining symptoms to patients.

Part II. is reserved for treatment. Dr. Bousfield belongs to the increasing school of psychotherapeutists, who regard lengthy psychoanalysis after the manner of Freud as a major operation reserved for rare (and necessarily affluent) patients; the majority can be successfully treated by suggestion including hypnotism, mental reeducation, remedial exercises, brief psychoanalysis, drugs.

He does not despise the humble and ubiquitous bottle of medicine and believes in a full and complete physical examination.

The classification adopted is that of the psychoneuroses including conversion hysteria, obsessional hysteria, anxiety hysteria and the actual neuroses comprised of anxiety neuroses and neurasthenia. To these are added for practical purposes: (i.) Psychosexual disturbances of the sexual system as homo-sexuality *et cetera;* (ii.) combined organic and psychic diseases such as asthma *et cetera*; (iii.) deleterious methods of adjustment such as alcoholism.

One of the chapters especially worthy of close attention concerns disorders of menstruation and pregnancy. He rightly states that the use of the word "unwell" for a healthy physiological process is a pernicious suggestion productive of untold misery to women. Pernicious vomiting in pregnancy is often hysterical, rather than toxæmic and the reader is reminded that it often reacts favourably to psychotherapy. Under drug addiction he discusses the secret remedies and, whilst admitting his empiricism, believes injections of colloidal gold to have definite therapeutic advantages.

Dr. Bousfield is to be congratulated on writing a very readable book which requires no abstruse psychological knowledge on the part of the purchaser in order to be able to profit by its perusal, a commendable feature for the non-specialist.

[1] "The Pathology, Diagnosis and Treatment of Functional Nervous Diseases," by Paul Bousfield, M.R.C.S. (England), L.R.C.P. (London); 1926. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 224. Price: 6s. net.

## NORMAL PHYSICAL SIGNS.

IN the preface of his book "A Manual of Normal Physical Signs" the author, Dr. Wyndham B. Blanton, indicates that the purpose of the book is to aid the average student who, in studying textbooks of physical diagnosis, finds normal and abnormal signs so intermingled that he has difficulty in finding a clear description of normal physical signs.[1]

Since the scope of the book is limited, the student who uses it, will still have to unravel the tangle in the textbooks to which the author refers, and in addition the purchase of another book will be necessary. The descriptions of normal physical signs follow generally the usual teaching, but the manner of describing them differs.

The book is written in the notebook form and its brevity might appeal to students, but the study of lists of signs without diagrams might become monotonous. This applies particularly to the section on regional anatomy. It is advised that this skeletal arrangement should be amplified by classroom instruction. That is certainly essential. The notebook form might be useful to a teacher who cared to rely on notes prepared by someone else instead of preparing his own, but whether it is a desirable form for students is questionable. Normal physical signs of all the systems of the body are included and occasionally the author lapses and intermingles a few abnormal signs.

## LECTURES ON AURICULAR FIBRILLATION.

Two papers on auricular fibrillation, by J. G. Emanuel, of Birmingham, comprise the Ingleby Lectures for 1925.[2]

They give a useful survery of this important heart irregularity. Mackenzie's views of its nature are followed, but not to the entire exclusion of circus movement theories of Lewis and his collaborators. For example, auricular fibrillation is regarded as a form of abnormal rhythm in which the pacemaker has loosed its hold on the cardiac reins, its function being usurped by the ordinary fibres of the auricular walls. The well authenticated experimental evidence of circus movement receives rather cursory reference. The difference in the number of stimuli capable of traversing the bundle of His in normal rhythm and in auricular fibrillation is explained by the abnormal origin of these latter, arising in auricular fibres rather than from the sino-auricular node. But surely the refractory period of the auricular-ventricular node is the more important factor. The author believes that toxins act like digitalis on the bundle of His and produce slowing of the ventricle in cases of fibrillation; we cannot subscribe to this view, but can agree that chronic toxæmia from various causes often results in this irregularity.

It is pointed out that odd patients with auricular fibrillation may have persistent vomiting as part of the picture which is not due to large doses of digitalis, but is apparently a reflex stimulation of the vomiting centre by the rapid irregular heart action.

Of digitalis preparations preference is given to Nativelle's "Digitaline" granules 0·27 milligramme ($^1/_{240}$ grain) which he finds is more effective than two mils (thirty minims) of tincture of digitalis (British Pharmacopœia). Quinidine treatment is considered in detail and the various indications and contraindications are considered.

Altogether this is a valuable little book, published at a modest price and well worth its place in cardiac literature.

[1] "A Manual of Normal Physical Signs," by Wyndham B. Blanton, B.A., M.A., M.D.; 1926. St. Louis: The C. V. Mosby Company. Post 8vo., pp. 215. Price: $2·50 net.

[2] "Auricular Fibrillation," by J. G. Emanuel, B.Sc., M.D., F.R.C.P.; 1926. Birmingham: Cornish Brothers Limited. Royal 8vo., pp. 31. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 27, 1926.

## Aids to Diagnosis.

In every well appointed hospital there are many special departments where bacteriological, biochemical, immunological, radiological, electrical and other investigations can be carried out. In addition the patient can be sent to the neurologist, the dermatologist, the oto-rhino-laryngologist, the otologist, the ophthalmologist and the dentist for examination and report. The patient has the immense advantage in the hospital of the team work of the staff. In some institutions a thorough examination of all systems is conducted in all serious illness. Not infrequently this elaborate survey, undertaken without any special indications of a lesion of the several systems, reveals an unsuspected pathological process which may even be independent of the disease for which the patient is seeking treatment. Moreover, it is important from the point of view of the future well-being of the patient that a record is made that on a particular date no evidence of any abnormality of the systems examined was discovered. In private practice such an extensive examination is rarely attempted. To refer a patient to a dozen specialists would involve him in a very heavy expenditure which only a rich man could afford. It may be asked whether so searching a study is necessary for the correct diagnosis and treatment of an ordinary complaint. It is true that the diagnosis of many common diseases can be made from clinical signs and symptoms, but modern medicine demands more than the mere naming of the disease. If the practitioner wishes to have a clear picture of the condition of each organ in the body of the patient and to have a satisfactory conception of the manner in which the several organs are carrying out their functions, it is quite essential that every possible source of information must be exploited. It is always difficult and often impossible to ascertain exactly what is going on within the organism during the course of a disease. No one can afford to guess at a diagnosis when definite information can be obtained. At times the only means of diagnosis are those provided in the laboratory. In these circumstances the fullest exploration of every organ and of every fluid available should be conducted in all serious illness and in every obscure disorder.

The ideal remedy of the defect in the organization of the medical profession leading to the neglect of these manifold investigations in private practice would be the establishment of complete clinics like the Mayo Clinic where team work is provided. There are some obvious disadvantages connected with private clinics of this type. In the first place large and costly organizations tend to assume a commercial aspect. The institutions serve to advertise their owners and this inevitable advertisement may lead to other abuses. Private clinics of this kind cannot be established outside the cities, for the cost of upkeep is always considerable and failure will occur if the support be small.

The only other solution of the difficulty is to educate the public to the necessity of elaborate investigations and to induce medical practitioners to work in groups, so that the team idea can become a practical expedient. The general practitioner can contribute to the success of such an arrangement by equipping a small laboratory in his place of practice and by carrying out some of the investigations that do not demand the expenditure of much time nor the acquisition of much technical skill. We have claimed on a previous occasion that every general practitioner should be prepared to carry out the examination of blood as far as the enumeration of erythrocytes and leucocytes and the counting of the different forms of white corpuscles are concerned. The hæmoglobin estimation, too, can be undertaken. The technique of blood sugar estimations can be learned without prolonged training in chemistry and is worth while acquiring. The same may be said of the quantitative examination for urea in the blood. Test meals and the examination of the gastric contents should be within the scope of the general practitioner's undertakings. He can carry out many of the simpler bacteriological investigations, although such procedures as the isolation of bacteria in pure culture and the study of their reactions are certainly too time robbing to be enter-

tained by the busy practitioner. The majority of the immunological tests demand constant practice for reliable results and on this account they should be referred to the immunologist. On the other hand those biological tests which are carried out by introducing an antigen into the skin of the patient, are easily performed by the busy practitioner. The testing of urine and of fæces, even when the fullest information is required, should be regarded by the general practitioner as his work, at all events in the first instance. This includes quantitative estimations of albumin, sugar, chlorides, urea, non-protein nitrogen and certain abnormal constituents and the qualitative tests for blood, pentose, hemi-albuminose, acetone and many other bodies. If some of these investigations were undertaken by the general practitioner in his own small laboratory, the number of patients who are referred to the special pathologist, would be materially reduced. It was claimed on the previous occasion when this suggestion was made to general practitioners in these columns, that the proposal imposed too heavy a demand on the time and special knowledge of the practitioner. The reply to this criticism is that many general practitioners have done all this. They have taken the trouble to acquire the necessary technique; they have expended a small amount of money for the requisite apparatus and have become so fascinated in the work that they were not too tired on their return after a heavy day to spend a short time at the work. Given the ability to do it and the will to become efficient at laboratory work, even the busiest man will be able to steal an occasional hour.

## Current Comment.

### PNEUMONIA.

ACCORDING to G. W. Norris and D. L. Farley, writing in the latest edition of "Osler's Modern Medicine," it is customary to regard pneumonia as a general pneumococcus infection with the lesion in the lung as but one of its manifestations. While this statement is true, it is worded in such a way that it may possibly be misleading. It implies that a generalized pneumococcal infection occurs and that the lesion in the lung is the result of that infection. It might be interpreted as suggesting that a pneumococcal septicæmia brings about an aggregation of pneumococci in the tissues of the lung and that they, finding the nidus suitable, multiply and produce the clinical picture of pneumonia. This thought immediately presents the question of the definition of the term septicæmia. Septicæmia means more than the presence of microorganisms in the blood. If what may be described as overflow pneumococci are washed into the blood stream from a consolidated area of lung, the defensive powers of the lung are generally sufficient to deal effectively with them. This condition should not be regarded as septicæmia. When the bacteria, however, persist and multiply in the blood, a septicæmia is present. An infection of the body by the pneumococcus produces a lesion in some area—lungs, peritoneum, the synovium of a joint, the meninges—and the lesion becomes a focus for the continued development of the pneumococci. A septicæmia may or may not be superadded.

It is not necessary to do more than refer to the well known fact that pneumococci may be found in the mouth and pharynx in large numbers of the population. It is obvious that for the production of pneumonia virulent pneumococci must gain access to the lung. Two views are held as to the manner in which the lung becomes affected. According to the first the cocci are carried from the portal of entry to the lung by the blood stream and according to the other the cocci are carried by the air into the bronchioles and alveoli, find lodgement there and attack the tissues of the lung directly. The latter is the view most generally accepted and this is owing largely to the experimental work of Blake and Cecil in 1920. These workers produced pneumonia in monkeys by intratracheal injection of pneumococci which were highly virulent for mice.

The experimental production of pneumonia in monkeys has again been studied by Dr. Otto Schöbl and Dr. A. W. Sellards.[1] They followed closely the technique of Blake and Cecil. They point out that the degree of virulence of the pneumococcus is an important factor. Blake and Cecil used cultures of such virulence that 0·0000001 cubic centimetre of a broth culture would kill a mouse within forty-eight hours of intraperitoneal injection. Type I. pneumococci were used and inoculations were made after cultures had been incubated for eighteen to twenty hours. A uniform volume of one cubic centimetre of fluid was injected by direct puncture through the skin into the lumen of the trachea below the larynx. The degree of virulence was maintained by the passage of the stock strain through mice twice each week during the course of the investigation. As a preliminary step thirteen stock monkeys were examined for pneumococci. A small amount of saliva was injected into the peritoneal cavity of white mice. Cultures were then made from the heart blood of the mice. From eleven of the thirteen an abundant growth of a Gram-positive coccus producing green pigment was obtained. Three of these strains of cocci proved to be pneumococci. The occurrence of pneumococci

[1] *The Philippine Journal of Science*, September, 1926.

in the mouths of healthy monkeys is held to explain some of the unexpected results obtained by Blake and Cecil. These workers found that healthy monkeys when placed in contact with monkeys suffering from a Type I. pneumonia, became infected with pneumonia due to Type IV. pneumococcus. Dr. Schöbl and Dr. Sellards regard it as unnecessary to accept the view of mutation of type put forward by King in this connexion. Nineteen monkeys received intratracheal injections of pneumococci and ten of these became affected by pneumonia. It is interesting to note that in one monkey that was killed at the end of the second day, no pneumonia was present, but one colony of pneumococci was cultured from one cubic centimetre of blood. Six monkeys were given intravenous injections. At autopsy acute lesions were found in many parts of the body, but no pneumonia was present. Six monkeys were given subcutaneous injections of pneumococci. Four remained well, one became ill and recovered and the other died during the first day from septicæmia. Intravenous injection of pneumococci was combined with intratracheal injection of sterile broth and of killed cultures of pneumococci. This was done in order to determine whether the presence of material which was more or less irritating to the alveoli, would influence the localization of pneumococci in the presence of a septicæmia. All such experiments were without significant effect.

Dr. Schöbl and Dr. Sellards describe the macroscopical and the microscopical appearances in the lungs in which pneumonia was produced. The macroscopical appearances were at first sight suggestive of lobar pneumonia, but it was found that the apparent confluence of the hepatization was due to extensive œdema and created this impression. In no case was real red or grey hepatization found to involve either the major part of the lobe or the entire lobe. The microscopical appearances of the areas of grey and red hepatization were similar to those usually found in these conditions. The most pronounced hepatization was found at the hilus of the lungs, particularly on the right side, corresponding with the first and also the second branch of the right bronchus.

In discussing the pathogenesis of this experimental pneumonia Dr. Schöbl and Dr. Sellards point out that pneumonia occurred in no instance after intravenous injection of the culture alone or after intravenous injection of the culture combined with intratracheal injection of sterile material. They conclude that the pathological changes in the lung are not of hæmatogenic origin. The intratracheal injection was the only one followed by pneumonia. The situation of the lesions in the lung can be explained by the anatomical conditions of the bronchi. Injections were made while the animal was lying on its back under general anæsthesia and it was thus natural for the fluid to run straight into the right bronchus and produce a centrally situated area of pneumonia. From the microscopical appearances the impression was gained that no matter how extensive the change in one and the same lobe might be, successive changes of propagation were always noticeable; the changes had taken place successively rather than simultaneously. Evidently the infection followed the same paths as are taken by inert bodies. Cellular infiltration was present in the peritracheal tissue and in the tissue surrounding the large and small bronchi in affected areas and it was concluded that the inflammatory process commenced in the bronchi, gradually involved the peribronchial tissue and followed the lymphatic spaces. While the peribronchial infiltration and the interstitial infiltration of the alveolar septa are due to the intratracheal or intrabronchial invasion, the diffuse engorgement of the capillaries, present over the entire lung, is due to the septicæmic stage of the infection. Moreover, vascular changes of this nature are described in nearly all the protocols of experiments carried out by these observers by intravenous injection. It may thus be concluded that the inflammatory process resulting from intratracheal injection spreads in two ways, directly by the alveolar passages and also by the surrounding lymph passages. Dr. Schöbl and Dr. Sellards point out that the striking feature of experimental pneumonia in monkeys is this perivascular and interstitial inflammation and that this feature is not pronounced in human lobar pneumonia. They hold that their demonstration of the extension of the inflammatory process by the perivascular lymphatics and the alveolar septa confirms the finds of Blake and Cecil. Unlike the latter, however, they regard the perivascular and interstitial changes as the secondary rather than the primary process. They look upon the peribronchial lymphangitis of the large and small bronchi as the condition responsible for the state of septicæmia rather than as a great factor in the pathogenesis of experimental pneumonic lesions.

The differential points between bronchopneumonia and lobar pneumonia in man are usually stated to be quantitative and qualitative. The quantitative differences refer to the extent of the lung tissue involved. The qualitative differences are that the hepatization of lobar pneumonia is simultaneous in the same lobe, while in bronchopneumonia the hepatization is characterized by the successive formation of foci in the same lobe. It is frequently difficult to differentiate between confluent bronchopneumonia and lobar pneumonia. Dr. Schöbl and Dr. Sellards express the opinion that the view is becoming more generally accepted that the difference between the two types of pneumonia depends on the virulence of the pneumococcus on the one hand and on the resistance of the animal body on the other. The balance between the invading organism and the resistance of the host may swing to one side or the other. Thus they conclude that in man infections with the pneumococcus may result merely in a carrier state or in bronchitis, in bronchopneumonia with septicæmia or in typical lobar pneumonia with septicæmia. The difference between lobar pneumonia and bronchopneumonia is thus merely one of degree and the statement of Norris and Farley to which reference has been made, agrees with this conception.

# Abstracts from Current Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### The Use of Immune Goat Serum in the Prevention of Measles.

RUTH TUNNICLIFF AND ARCHIBALD L. HOYNE (*Journal of Infectious Diseases*, January, 1926) consider that in view of the results of their investigations into the prevention of measles by the use of immune goat serum further studies on this subject are indicated. Tunnicliff has described the isolation from the blood of a filter-passing, Gram-positive, green-producing diplococcus almost constantly in the early stages of measles. The organism found was recovered in smear preparations and in cultures from the respiratory tract. Specific agglutinins, opsonins and complement fixing bodies for the coccus have been demonstrated in the blood of patients with measles and lesions and symptoms have been produced in animals by the organism. Rabbits inoculated with the coccus manifested no reaction when reinoculated with fresh measles material. In view of these findings an attempt was made to produce immune serum with the organism in large animals; the goat was selected rather than the horse on account of sensitization to horse serum, if the patient had previously been treated with diphtheria antitoxin. The serum of a goat inoculated with the diplococcus protected rabbits against a subsequent injection of infective material from patients suffering with measles. Control rabbits injected with normal goat serum and those untreated manifested some or all the characters of measles in rabbits, raised temperature, Koplik spots and an eruption. Serum from convalescent goats was found to protect human beings against measles as effectively as serum from convalescent measles patients.

### Researches in Sprue.

N. HAMILTON FAIRLEY AND F. P. MACKIE (*The Indian Journal of Medical Research*, July, 1926) give a progress report on their researches in sprue. In studying the ætiological aspect of sprue they first turned their attention to the association of certain species of Monilia with the disease as emphasized by Ashford. They isolated and studied one hundred strains of yeasts from one hundred and eleven cases of sprue and other diseases and found that Ashford's strain was recovered in about half the cases of sprue but in non-sprue conditions and in healthy persons to a similar extent. They failed to confirm the findings of Martinez and Michael who reported a complement fixation in the sera of sprue patients using extract of *Monilia ashfordi* as antigen. The authors concluded that the Ashford strain may be regarded as a secondary factor in the production of certain intestinal features of sprue, but never as the primary cause. In investigating the aerobic intestinal bacilli in cases of sprue it was found that to a certain extent they resembled those of dysenteric stools. In studying the pathology of sprue it was noted that the inflammatory lesions in the intestine varied greatly. In some cases a subacute inflammatory process characterized by injection and congestion of vessels, superficial exudation and desquamation of surface cells was noted; in others minute ulcers were discovered and in others again gross ulceration leading to perforation and death was noted. The tongue condition varied also. Atrophy of the fungiform and filiform papillæ was seen in two cases at autopsy. Other viscera were affected by atrophic changes only. The pancreas was never found to be diseased; the spleen was constantly fibrosed and generally pigmented, but these changes were possibly associated with chronic malaria. No changes in thyreoid or suprarenals were found. The bone marrow presented a complete aplasia in some cases and in one presented the picture of pernicious anæmia. Fractional test meals were carried out in twenty-six cases of sprue and hypoacidity or actual achlorhydria was noted in fifteen. The importance of these findings is stressed by the authors, since they show that the fractional test meal enables the observer to differentiate between sprue and pernicious anæmia. In the latter complete achlorhydria exists due to a secretory defect, in the former the hydrochloric acid is secreted efficiently, but becomes neutralized by alkaline secretion from the duodenum. In a series of twenty-five consecutive cases in which blood counts were done, the average count was: Red cells, 3,243,100 per cubic millimetre; hæmoglobin value, 65·1%; colour index, 1; poikilocytosis, anisocytosis and polychromasia were frequently present, but nucleated cells, especially megaloblasts, were rare. The white cell counts revealed leucopenia, the total count in a consecutive series of twenty-four cases was 6,367 per cubic millimetre. Decrease of neutrophile cells and increase of lymphocytes are usual. Van den Bergh's reaction was found to give low readings (indirect reaction) in sprue while high readings are the rule in pernicious anæmia. As a result of combined clinical and pathological observations the authors state that they regard sprue "as due to some infective agency primarily involving the mucosa of the alimentary tract and leading to the generation of toxic substances which deleteriously affect the blood elements, bone marrow and parenchyme cells of the internal organs."

### The Dysentery Skin Reaction at Different Ages.

H. BROKMAN AND S. POPOWSKI (*Journal of Immunology*, July, 1926) refer to their previous report on the skin reaction which follows intradermal injection with the toxin of the Shiga-Kruse bacillus, the reaction depending on the absence or presence of antitoxin in the blood. They give their findings in the dysentery skin reaction at different ages. They examined six hundred and seventy-five individuals and divided them into three groups: (i.) children up to three years, (ii.) children from four to sixteen, (iii.) persons from sixteen to twenty-three. The amount of toxin introduced was one-hundredth of a lethal dose per kilogram of rabbit. In Group I. reactions were obtained in 51%, in 27% in Group II. and in 59% in Group III.

### An Anomaly in Blood Groups.

REUBEN OTTENBERG AND ALICE JOHNSON (*Journal of Immunology*, July, 1926) give details of a hæmolytic accident following a blood transfusion and discuss the dangers of the use of universal donors. The patient was a woman, aged fifty-one years. Illness commenced with severe hæmorrhage from the nose a year before admission to hospital. Ascites and greatly enlarged liver and spleen were present. The red cells numbered 2,600,000 per cubic millimetre, the hæmoglobin value was 50%, the white cells numbered 3,000 per cubic millimetre; neutrophile cells were 53%, lymphocytes 54%, basophile cells 1% and large mononuclear 2% of the total. The serum failed to yield a reaction with the Wassermann test. Large urobilin excretion and high bilirubin index in blood plasma were present. With a view to improving the patient's condition so that splenectomy might be carried out, a blood transfusion was performed. The donor belonged to the same group as the patient, Group III. Five hundred cubic centimetres of blood were administered by the Unger method. Soon after the transfusion the patient had a rigor which lasted for six hours and eight hours later was observed to be jaundiced. Vomiting with red cells and hæmoglobin in the urine followed. On the second and third days bile appeared in the urine. Six days after the transfusion the patient died. The day of her death the red cells numbered 1,180,000 per cubic millimetre and the hæmoglobin value was 22%. At the autopsy the liver was not enlarged, but the cells in the central zone were atrophied and in places had completely disappeared. The spleen was enormously enlarged. On retesting the donor's blood the report that it and the patient's blood belonged to Group III. was confirmed, but the donor's serum was found to agglutinate the patient's red cells. Further testing showed that the donor's serum agglutinated the cells of nearly all other persons tested whether belonging to Groups I., II. or III. The donor was a so-called "professional donor" and had been used for blood transfusions fifteen or twenty times in six or seven years. He recalled one previous accident following transfusion with his blood. The authors assume that the hæmolysin introduced in the donor's blood accentuated the hæmolytic process

already going on in the patient's body and produced an acute failure of liver function. They suggest that so-called "universal donors" are not always safe, that hæmolysis is particularly dangerous in diseases of the liver and spleen and that cross tests of donor's and patient's serum and red cells should invariably be carried out to avoid the danger due to subgroups or rare anomalies.

### Kahn's Test in Leprosy.

M. V. ARGUELLES (*The Phillipine Journal of Science*, July, 1926) reports the result of an investigation of the Kahn test in leprosy. He found that both the Wassermann and Kahn tests did not yield reactions in a large majority of leper patients. In 1% samples of serum from lepers and from those suspected of leprosy Wassermann reactions were obtained in the absence of any signs or history of syphilis or yaws. In the serum of confirmed lepers a reaction to the Wassermann test was obtained in 1·23% of cases without signs or history of syphilis or yaws. The Kahn test yielded a reaction in 5% of leper and suspected leper patients without history or signs of syphilis or yaws. In confirmed cases the Kahn test yielded a reaction in 6·17% of those without sign or history of syphilis or yaws. As far as lepers are concerned, the author found the Kahn test of greater value than the Wassermann test in excluding syphilis or yaws and of less value in establishing the diagnosis of these two diseases.

## HYGIENE.

### Non-Pulmonary Tuberculosis and the Public.

KENDALL EMERSON (*The Boston Medical and Surgical Journal*, May, 1926) approaches the subject of non-pulmonary tuberculosis and the public by asking two questions: "What does the public know about non-pulmonary tuberculosis?" and "How should that knowledge be increased?" He confines his remarks to bone and joint tuberculosis and points out that the public is receiving little information about non-pulmonary tuberculosis through press reports. There is very little doubt, however, that with the splendid preventive measures now in vogue the relative number of cases of bone tuberculosis in a given community has materially diminished. He maintains that there is a danger that public attention may wander too soon from the gravity of the situation and that mothers may grow incautious and not seek early and persistent treatment for children with incipient evidence of disease in their joints. If such a state of affairs exists, the medical profession is then faced with the query: "What educational measures are necessary?" Medical men acquainted with the dread menace of tuberculosis when it attacks growing joints have a clear duty to perform. It is their duty to preach to a forgetful public the need of a proper degree of solicitude for suspected joints in children. The educational responsibility of the individual physician is not lessened by the establishment of splendidly equipped and up-to-date institutions where these conditions can be successfully treated.

### Tuberculosis and Employment.

A. NEVILLE COX (*Journal of State Medicine*, May, 1926) points out that for certain tuberculous patients all will admit that the colony system as realized at Papworth is admirable and that it is probably the best solution of the problem that can be attained. Such patients, however, form a very small proportion of the total tuberculous population. Thus Papworth and its like cannot be expected to solve the problem of employment for more than a few patients. What of the rest? There are only two proper alternatives—either the tuberculous worker must receive a pension or better still his working capacity must be used and rewarded for what it is worth. It is a fact, however, that a majority of the tuberculous patients are actually at work in spite of their disability and in spite of the state of the labour market. The author has inquired into the condition as to employment of six hundred and fifty-seven tuberculous patients under observation from the Brighton Dispensary and the results go to show that roughly 60% of the men and 70% of the women are at work in various occupations. He will not say that all these are working at suitable jobs and he points out that the dispensary does its best to dissuade patients from following an occupation when there is a risk of infecting children or when they will have to handle food after cooking or food that is eaten raw. While discussing the bearing of infection risk on employment the author asserts that there is a real danger in a tuberculous person looking after babies or children or working in close association with young adults. There is a smaller risk in a consumptive handling food, but apart from these there is no work which a patient may not safely do. Not every patient with tuberculosis is infectious or infectious in the same degree and yet there are workers who will give the cold shoulder to one of their fellows who has the misfortune to have tuberculosis, however careful he is, but will tolerate promiscuous spitting about a workshop by alleged healthy people. Many occupations are unsuitable from the individual's point of view. Occupations requiring great physical strain or good breathing power or occupations that are very dusty, cannot usually be followed, but every tuberculosis officer knows of patients working and working successfully at occupations apparently quite unsuited. There are four possibilities for a patient after discharge from sanatorium: (i.) The disease may be advancing and employment is out of the question; (ii.) the patient may think he can return to his occupation after a few weeks spell; (iii.) he may return to work and break down again; (iv.) he may return to work and continue at it successfully for years. It is with the two middle classes that the author is concerned. There is abundant evidence that if the tuberculous patient can weather successfully the two years after his discharge, he may survive for many years in moderately good health and with a moderate working capacity. During this period there are many patients unfit for the ordinary work-a-day world, but fit for a few hours' work each day and the better for it mentally and morally. Local authorities and government departments should give a lead to private employers in accepting this type of tuberculous person for employment.

### The Seaman's Health.

F. HUMBERT (*The World's Health*, June, 1926) points out that one category of workers who have benefited very little by the recent advances in industrial hygiene is that of the seamen. He does not refer to shipping companies which have medical officers on board their ships, but points out that there is a large number of seamen for whom no medical examination or supervision is provided. These receive no warning of impending danger and no regular health teaching about the risks of special diseases. Their quarters on board are necessarily crowded and their food monotonous. Their hours of work and rest are very irregular owing to the unforeseen play of the elements, while cargo and bilge water often disseminate microbes or produce asphyxiating gases which spread over the ship. Although the sailor spends part of his time in bracing sea air, the rest of it he is obliged to spend in an atmosphere which is stifling owing to faulty ventilation. The fireman's job is particularly unhealthy and disagreeable. Seamen, being obliged to live temporarily in such close quarters, are more dependent on one another than other workers and the danger of contagion is increased. Besides this, they are so careless of their health and place so much confidence in their natural resistance that they may easily embark carrying with them the germs of some disease like tuberculosis which will develope on board. In the Norwegian merchant service in 1900 there were 453 deaths, 212 sailors were drowned, 153 died from unknown or what the author describes as "unrecognizable" causes, while 47 were accidentally killed. The author holds that something should be done and since it is impossible always to have a doctor on board, suggests: (i.) Educational propaganda on board ship by posters, pamphlets and other means; (ii.) improvement of existing manuals on hygiene for seamen or publication of new ones; (iii.) the establishment of health centres with clinics for the medical treatment of seamen; (iv.) the drawing up of plans for the supply of up-to-date standardized medicine chests.

# British Medical Association News.

## MEDICO-POLITICAL.

A MEETING of the members of the Queensland Branch of the British Medical Association resident in the West Moreton area was held at Ipswich, Queensland, on October 16, 1926, for the purpose of inaugurating the West Moreton Medical Association in affiliation with the Branch.

A motion proposed by DR. J. A. CAMERON, seconded by DR. BROWN, to form a West Moreton Medical Association was unanimously carried.

The following officers were elected:

*President:* Dr. J. A. Cameron.

*Honorary Secretary and Treasurer:* Dr. G. H. Brandis.

*Committee:* Dr. D. E. Trumpy, Dr. E. Elmslie Brown (Ipswich), Dr. Ethel Orchard (Esk), Dr. Robert Wallace (Rosewood) and Dr. W. A. Fraser (Boonah).

It was decided to hold two regular meetings a year in October and April respectively.

A discussion took place on the hospital question and lodge matters.

# Medical Societies.

## THE ALFRED HOSPITAL CLINICAL SOCIETY.

THE ANNUAL MEETING OF THE ALFRED HOSPITAL CLINICAL SOCIETY was held at the Alfred Hospital, Melbourne, on September 28, 1926, DR. M. D. SILBERBERG in the chair.

### Endocrine Disorder.

DR. SILBERBERG presented a patient suffering from endocrine disorder. She was a girl, thirteen years of age, weighing one hundred and eight kilograms (seventeen stone). The skin manifested *striæ albicantes*, but was not particularly coarse. The axillary and pubic hirsutes were well developed. The systolic blood pressure was 130 millimetres of mercury with a diastolic reading of 70 millimetres. The basal metabolic rate estimation proved to be −18. Menstruation had commenced at the age of ten and had been normal. The visual fields, the fundi and the blood sugar curve presented no abnormalities. A skiagram of the *sella turcica* revealed nothing unusual. The limits of sugar tolerance had not been tested.

She had slowly gained in weight from one hundred to one hundred and eight kilograms (about sixteen to seventeen stone) in the seven months she had been under observation in spite of treatment with various gland combinations. She had been given thyreoid extract up to 0·38 grammes (six grains) *per diem*. She had had daily injections of pituitrin. Ovarian and parathyreoid glands had been tried.

DR. S. LAURIE in discussing this case remarked that the condition was certainly not a Fröhlich's syndrome. He would call it hypergenitalism, in boys it was said to be due to a pineal tumour, in girls possibly to ovarian disturbance. He had had one patient with similar characteristics. She had presented the same mature manner and appearance, that of a girl far older.

DR. H. C. COLVILLE suggested intravenous glandular therapy; DR. W. L. CARRINGTON suggested the use of thyreoxin and DR. W. S. NEWTON, Armour's thyreoid extract.

### Vasomotor Disturbance.

DR. W. S. NEWTON discussed the vasomotor disturbance in a woman of thirty years. For twelve months past the left hand had been painful and colder than its fellow. It manifested a blue colouration with a slow restoration of the circulation after pressure. In every other way the patient was in good health. The onset had been marked by pain and stiffness of the fingers and coldness. The condition had remained unchanged ever since its onset in spite of many forms of treatment. An operation had been suggested consisting of a stripping of the brachial artery of its surrounding small nerves. Dr. Newton called for comments upon such a procedure and for any other suggestions as to treatment.

DR. J. F. MACKEDDIE, in discussing this case observed that no neurological abnormalities were to be detected. He nevertheless felt that there was a definite functional element present and advised that the hand be no longer carried in a sling and that the patient be urged to use it freely and given every encouragement and hope.

### Paget's Disease.

DR. J. R. ANDERSON presented a patient suffering from Paget's disease who had been under his observation for a considerable period. She was a woman of about forty-five years and the onset of the disease had occurred some eighteen months previously. Her head had steadily enlarged and a heavy boss of bone was present on each side just above the ear. Her height had decreased, her lower limbs had bent outward at the knee and her vision had been affected. Several X ray films were exhibited, showing patches of rarefaction coexistent with areas of condensation of bone throughout the skull. The Wassermann test had yielded no response.

### Ulnar Nerve Paralysis.

MR. C. J. O. BROWN showed a patient whose ulnar nerve paralysis had first been noticed four months after a subglenoid dislocation of the shoulder. The patient was a woman aged forty-six years who had fallen on her outstretched hand and dislocated her right shoulder; at the time of the accident she was found to have a musculo-spiral nerve paralysis. This had been treated on a cock-up splint after reduction of the dislocation and four months later she returned with typical rigid *main en griffe*. Examination revealed complete loss of epicritic and protopathic sensation over the area supplied by her ulnar nerve. Deep sensation was normal. All the small muscles of her hand supplied by the ulnar nerve were paralysed, atrophied and contracted. The *flexor carpi ulnaris* and the deep flexors of the ring and little fingers were functioning normally. X ray examination revealed small pseudocervical ribs on both sides, but the typical ulnar distribution of the signs excluded this diagnosis.

The condition was diagnosed as an incomplete ulnar nerve lesion occurring at the time of the dislocation and overshadowed by the more obvious musculo-spiral paralysis.

# Obituary.

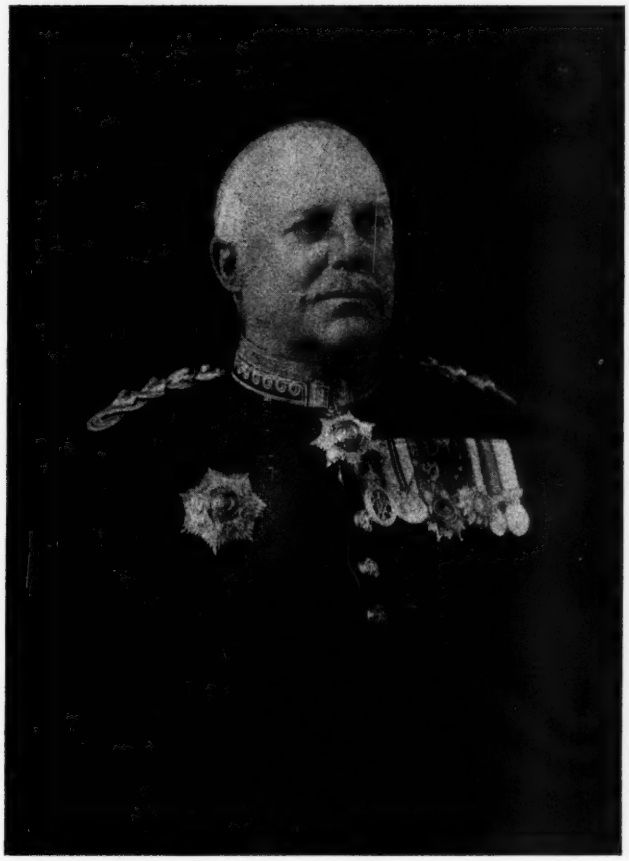
## CHARLES SNODGRASS RYAN.

To have gained a reputation during many years as a sound and successful surgeon, to have attained eminence as a soldier, to have been respected and admired as a teacher and to have been beloved and honoured as a colleague is a record which few may claim. That these attributes may justly be ascribed to Sir Charles Snodgrass Ryan whose death was recently announced in our columns, secures for his name a place among the leaders of Australian medicine.

Charles Snodgrass Ryan was the son of the late Charles Ryan, of Derriwait, Victoria. He was born in 1853 at Longwood, Victoria, and at the age of ten years was sent to the Melbourne Grammar School. After some years of study at this school he realized that medicine was to claim his attention and he went to the other side of the world to pursue his objective. Edinburgh was the school of his choice. With the energy which characterized his actions in after life, he made the most of his opportunities and graduated as bachelor of medicine and master of surgery in 1875. Before taking up practice he went to Bonn and Vienna for post-graduate study. It was at the latter place that Ryan first heard the call of the God of War, the call which few men in the heyday of health and vigour can resist.

There can be no doubt that Ryan's subsequent career was influenced in no small degree by his military activities.

The soldier's first and indeed only consideration must be his faithful and unswerving performance of duty in the face of personal danger. The man is a unit in the fighting machine and the safety of the machine must be the primary consideration. Ryan's character gave evidence of this training. In later years what many an Australian "digger" regarded as harshness, was an inflexible performance of duty as he saw it, duty carried out in spite of personal feelings and personal loss. Ryan's military career began with the Turko-Servian war in 1876. He volunteered as medical officer in the Turkish army and he also served on the Turkish side in the war between Turkey and Russia. His presence for more than four months at the siege of Plevna earned for him the *sobriquet*, Plevna Ryan. He escaped capture at Plevna by being sent away in charge of wounded Turkish soldiers. He also went through the seige of Erzeroum. Subsequently he was captured by the Russians and held as prisoner of war. For his services to Turkey he received the Fourth Order of the Osmanieh and the Fourth Order of the Medjidie. He published an account of his experiences with the Turkish army in a book entitled, "Under the Red Crescent."



It was after this his first experience of war that Ryan came to Melbourne. His surgical ability soon asserted itself and he became attached to the Melbourne Hospital and to the Children's Hospital, Cartlon. He served these institutions faithfully and well and after many years of active work became consulting surgeon to both. His influence was widespread and bore fruit in the surgical equipment of many generations of students who passed through his hands. Nothing was too much trouble to him; he extended the same courtesy to a student from another State as he did to a visiting surgeon. Hence it was natural that his personal friends as well as his admirers were numerous. He took an active part in the affairs of the Victorian Branch of the British Medical Association. He became President of the Medical Society of Victoria in 1893 and Trustee of the Society in the same year. The latter position he held until he left for his last trip to Europe.

At the outbreak of war in 1914 Ryan might well have sat back and have left the work to younger men, but the military experience of earlier years could not be quenched and he insisted on going abroad with the Australian Imperial Force. His first appointment was that of Assistant Director of Medical Services to the First Division. In this capacity he landed in Egypt in 1914. It was not long, however, before he was attached to General Bidwood's staff and thus it came to pass that at the landing on Gallipoli he opposed the soldiers of the country with whom he had fought thirty-seven years before. It is said of him that he did not expect to survive the campaign on Gallipoli and this nearly was so, for he was stricken with enteric fever in a severe form. It was months before he completely recovered. After his convalescent period was over he again "carried on" and as consulting surgeon at Horseferry Road he dispensed orders of "return to Australia" or "fit for duty" with an impartial hand. For his services in the war he was first created a Companion of the Most Honourable Order of the Bath in 1916 and later received the honour of knighthood as Knight Commander of the Most Excellent Order of the British Empire. He was also a Companion of the Most Distinguished Order of Saint Michael and Saint George.

Ryan had other interests outside his professional work. For many years he was consul of Turkey in Victoria and he was a member of the Council of the Royal Zoological and Acclimatization Society and he also held office for a time as President of the Old Melburnians. Of his character and of his personal qualities Sir George Syme and Mr. Hamilton Russell are best qualified to speak. Their tributes to his memory are appended.

With the pasisng of Charles Snodgrass Ryan Australia loses one whose memory will be cherished for what he was as a man and for what he gave to the community. His devotion to duty and his fixity of purpose will serve as an example to those who come after him.

Sir George Syme writes:

It was my privilege to have known Charles Ryan intimately ever since he returned to Melbourne from the Russo-Turkish War—during my student days. He appeared then as a hero of adventure and romance and his *debonnair* manner made him universally popular.

I well remember his first operation at the Melbourne Hospital. The theatre was crowded to see the surgeon of Plevna at work. He was rather taken aback and nervous, but came in jauntily whistling a popular air. Charles Ryan was elected surgeon to in-patients at the Melbourne Hospital in the same year that I became his corresponding surgeon to out-patients and I assisted him at operations both in the hospital and in private practice. When he went trips to Europe, I took his place at the Melbourne Hospital and as Medical Officer of Railways and carried on his private practice. I thus came to know the high esteem in which he was held by all sections of the community.

"Charlie" Ryan, as everyone called him, was universally beloved, because he had such a generous, kind heart, such genuine sympathy and such a buoyant, genial temperament. He came into a patient's room or a hospital ward like a cheering sunbeam.

Besides being associated with him in his professional work it was also my privilege to spend many week-ends and holidays with him at his country house at Frankston and to accompany him on several trips to country parts of Victoria, to New Zealand and to Luxor, in Egypt, the last

we had together. He had a boyish enthusiasm that charmed everyone and made him new friends wherever he went. He was very interested in natural history. I have gone bird-nesting with him which he enjoyed as much as any schoolboy. He had a great collection of birds' eggs and of birds which he had stuffed himself, for he learned taxidermy. He was fond of all animals and of all kinds of sport, especially shooting and fishing and also found delight in the beauties of natural scenery of which he took excellent photographs. Just before he left on his last visit to Europe he spent an evening with my family, showing us his numerous coloured photographs and telling stories in connexion with them. He was an excellent *raconteur* and had travelled so much and had met so many interesting people that his conversation was most entertaining, spiced as it was with his Irish wit. He had also read much and widely, not only current professional literature, but history, biography and travels. He had a remarkably retentive memory and very shrewd judgement. He formed opinions very quickly and carried them out rapidly, so much so as to give the impression to those who did not know him well, of being superficial and perfunctory, but, though rapid, he was really very sound. In the same way his operative methods might appear rather "slap-dash" to some, but he got most excellent results.

Charles Ryan was a man who lived his life to the full. He worked hard and enjoyed the pleasure of life fully as well. He had a special gift for friendship and was loyal and generous to the last degree, helping his friends to share in his own joy in life.

Mr. R. Hamilton Russell writes:

I can think of no man who will be more missed in Melbourne than Sir Charles Ryan. For us, his friends, the title conferred upon him by the King can never displace nor seem quite so fitting as that by which he has been affectionately known for so many years among his own people; so for us he will always be just "Charlie Ryan" of cherished memory. Those of us who were for a long time in intimate association with him in his work would soon come to recognize the presence of a native ability and forcefulness which would often carry him far towards the goal of efficiency that another with more showy intellectual qualities would fail to reach. In character he was unique; I have never known a man quite like him and I had good opportunities for observing him in days gone by when I was closely associated with him in both hospital and private work. Never was a nature more sunny and never was the borderland more narrow betwixt laughter and tears. One was always either laughing with him or smiling at him, but whatever the jest might be, it was never one that had been better left unspoken.

There is much that could be written about so vivid a personality as Charlie Ryan. One should speak first of his chivalry towards women and towards the weak, of his almost childlike simplicity and good nature and of even more significance, his intolerance of whatever conveyed the least suggestion of deviousness or unfairness. He would travel by no road that was not rigidly straight.

There was no doubt that Charles Ryan was a very brave man, brave with the physical courage the soldier prays may be his when the moment of test comes. In the Turkish army of 1877 he is said to have attracted no small amount of notice by reason of personal valour and even in civil life is there not a story of a maniac who started revolver practice in a theatre and of the promptitude and reckless courage of Ryan who leapt over the seats in order to come to grips with an armed madman? Even his little peculiarities were such as to make him the more lovable. His curious habit of making presents of all sorts of things of personal or domestic value would be embarrassing at first; later, one discovered the correct method of dealing with the situation, which was to pretend to accept the gift with due acknowledgments and no fuss, after which the episode might be regarded as closed and presumably forgotten by both parties.

He dearly loved "society" and was greatly sought after, for no matter what the gathering might be, his presence would go far towards making it cheerful and happy. Was there anyone else who could be at once so naïvely self-confident, so easy and charming in his manners and so amusing? He had a genius for friendship and his friends were cosmopolitan. The number of friends he possessed in his own land was made evident in the very large gathering that assembled in Saint Paul's Cathedral and at the graveside in the Melbourne General Cemetery on October 27 when his earthly remains were laid among those of his own family.

## WILLIAM ARTHUR HANDCOCK BURKITT.

THE record of a man's life in cold words in a printed page often conveys to strangers little of his real personality. Even when the description is skilfully and artistically

composed, the pen picture usually lacks the many minor characteristics, the typical gestures, the qualities of some predominating mood that are inseparable from the make-up of the man. It is feared that the account of the professional career of the late William Arthur Handcock Burkitt may fail to reflect in the minds of strangers an accurate picture of this sturdy and lovable son of Erin, as it may fail to satisfy those who knew and admired him.

William Arthur Handcock Burkitt was born at Kilkee in the parish of Kilfiera in the County of Clare sixty-three years ago. He was the son of the late Archdeacon G. Burkitt, an Irishman of English descent, who was known for his scholarship, his benevolence of thought and deed and his courage. The son was educated at Belfast and entered Trinity College, Dublin, at the age of twenty-one years. He inherited much of his father's erudition and ability. In the early part of his course he served under Traill and later he became prosector to Professor D. J. Cunningham. He attained the distinction of passing first in the senior division in anatomy, having gained 65% of the available marks. In his arts course he paid special attention to advanced mathematics. Throughout the later part of his student life he won the approval of his teachers and the golden opinion of his fellows. He was a keen Rugby football player and gained distinction in the field. He played for the University. His fine, massive frame, his endurance and muscular power gave him a great advantage over his contemporaries. In 1889 he took his degree in arts as well as the triple bachelor degree in medicine, surgery and obstetrics. After graduation he was appointed resident medical officer at the Adelaide Hospital, Dublin. Before he left Dublin his erstwhile teachers and chiefs gave him written evidence of their appreciation of his gifts and industry. He married in 1890 and immediately set sail for Australia in a sailing ship the *Rodney* as surgeon. The voyage in those days was neither luxurious nor even passably comfortable. But his splendid physique, his cheery optimism and philosophical bearing helped him to spend those tedious weeks happily. On his arrival in Australia he turned his face to the west, to the open country which he had learned to love in his youth. He did *locum tenens* work and insurance work in and about Bourke for a brief time and then dropped anchor at Goulburn. He acquired the practice of the late Dr. Gentle. From the start he was a highly successful general practitioner. He knew his work; he studied his patients; he brought the kindness of his nature, the sense of security that his bigness and gentleness combined awakened to all with whom he had professional dealings. Little wonder that he became as popular a doctor in Goulburn as he had been a student in Dublin. He employed no arts to draw himself close to those who sought his aid. The charm of his manner and his skill as a physician compelled men, women and children to seek him and to trust him implicitly. A year after his arrival in Goulburn, his son, now Professor A. N. Burkitt, was born. After six years he recognized that a refresher course would be of great advantage to his practice. His brother, Dr. Ormsby Burkitt, was available and agreed to look after his patients during his absence. He went to England and attended post-graduate courses.

About the year 1910 he found it necessary to put a brake on his professional activities. He had acquired some years previously a small station near Goulburn and in 1897 he bought about two thousand acres at Reevesdale. He was intensely fond of pastoral pursuits and found it difficult to refrain from the active management of this sheep and cattle station. In 1903 he bought two other stations, one at Tallong and one at Traralga. In spite of the work and responsibilities of his stations he attended to his practice with unabated zeal for all these years. He was a very sound obstetrician and his services in this branch of work were freely sought. He was fond of surgery and was a good operator. He was well and favourably known to his colleagues in Sydney and his reputation as a skilled family doctor spread during the course of years far and wide. After he had given up general practice with the exception of a few lodges in 1910, his opinion was sought in consultation by many of his fellow practitioners. In 1911 his wife died and in the following year he journeyed to England and America. During this trip he attended several post-graduate courses, for despite the fact that he had given up the greater part of his practice, he was always anxious to keep abreast of the times as far as his professional knowledge was concerned. He returned to Australia in 1913. When war broke out he was doing relatively little medical work. In 1901 he had joined the Australian Military Forces and had obtained a commission. He was appointed Medical Officer to the recruiting camp at Goulburn with the rank of Captain and did not spare himself in doing all he could for his country in its time of need. After the end of the war he devoted the greater part of his energies to the management of his stations. In 1925 he suffered from digestive disturbances and underwent an operation for gastric ulcer. He recovered well; he appeared again to enjoy the robust health that became his athletic appearance. He left Australia for England in the early months of this year, but suffered a recurrence of his previous trouble. In spite of all that could be done for him he died on September 7, 1926, a month after the second operation. The news of his death was quite unexpected by the majority of his friends and acquaintances and even his son and daughter were unprepared for the terrible shock.

William Arthur Handcock Burkitt had all the qualities that belong to the typical old-time family doctor. He was the friend of his patients; they came to him without a trace of misgiving and confided everything in him. His presence seemed to awaken confidence. He shared their woes when they were in difficulties and rejoiced with them when the fates were kind. He sought no public recompense for all he did. Indeed he hated loud applause. He was a keen sportsman and held many offices in connexion with sport. He was President of the Goulburn Racing Club, for he understood horses and owned some very fine racing horses. He played polo and was at one time President of the local polo club. He was President of the Goulburn Club where he was immensely popular. He also played a good game of golf. Men of his calibre are rare. The sympathy of the medical profession has been extended to Professor Burkitt and his sister.

# Naval and Military.

## APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 73, 78, 88, 90 and 97 of July 29, August 12, September 10 and 16 and October 7, 1926:

### PERMANENT NAVAL FORCES OF THE COMMONWEALTH—SEA GOING FORCES.

*Promotion.*—Surgeon Lieutenant William Edward John Paradice, M.B., Ch.M., is promoted to the rank of Surgeon Lieutenant-Commander. Dated 1st August, 1926.

### AUSTRALIAN MILITARY FORCES.

#### First Military District.

*Australian Army Medical Corps Reserve.*

Captain L. St. V. Welch is transferred from the Australian Army Medical Corps Reserve, 2nd Military District, 5th July, 1926.

Honorary Captain J. F. Merrillees is retired under the provisions of Australian Military Regulation 152 (1), 21st August, 1926.

Captain A. H. Baldwin is transferred from the Australian Army Medical Corps Reserve, 3rd Military District, 20th September, 1926; Lieutenant R. H. Fletcher, M.C., is transferred from the General List of the Reserve of Officers, and to be Honorary Captain, 1st September, 1926.

*To be Honorary Captains.*—James Brown, Harold William Horn, Herbert Spencer Roberts, Sydney Joseph McCafferty, Reginald James Nash, Claude Ewart Backwell, William Ellis George, Daniel Christopher McSweeney, Ralph Edward Weaver, Errol Auffere Cook, Charles Francis Anthony de Monchaux, Reid Innis Campbell,

Hugh Ward Anderson, James Alfred Broben, William Macfarlane Bevington, James Norman Main and John Leslie Dart, 6th September, 1926.

*Award of the Colonial Auxiliary Forces Officers' Decoration.*

*Australian Army Medical Corps.*—Captain W. J. Fearnley, Colonel A. H. Marks, C.B.E., D.S.O.

*Unattached List.*—Colonel D. G. Croll, C.B.E.

**Second Military District.**

*First Cavalry Division—Staff.*

Colonel J. C. Storey, O.B.E., V.D., Australian Army Medical Corps, is appointed Assistant Director of Medical Services, Divisional Headquarters, 1st July, 1926.

*Second Division—Staff.*

Colonel J. H. Phipps, D.S.O., V.D., Australian Army Medical Corps, is appointed Assistant Director of Medical Services, Divisional Headquarters, 1st July, 1926.

*Australian Army Medical Corps.*

*To be Lieutenant (provisionally).*—Charles Crawford McKellar, 3rd July, 1926. Lieutenant A. L. Watson, M.C., is appointed from the Retired List and to be Captain (provisionally), 30th June, 1926. Lieutenant-Colonel J. C. Story, O.B.E., V.D., is transferred from the Unattached List, 1st July, 1926.

*To be Colonels.*—Lieutenant-Colonel J. H. Phipps, D.S.O., V.D., Lieutenant-Colonel (Temporary Colonel) A. H. Tebbutt, D.S.O., V.D., and Lieutenant-Colonel J. C. Storey, O.B.E., V.D., 1st July, 1926. The provisional appointment of Captain P. L. K. Addison is confirmed.

Major (Honorary Lieutenant-Colonel) J. S. Purdy, D.S.O., V.D., is transferred from the Unattached List, 6th August, 1926.

*To be Lieutenant-Colonel Supernumerary to the Establishment of Lieutenant-Colonels, with Pay and Allowances of Captain.*—Major (Honorary Lieutenant-Colonel) J. S. Purdy, D.S.O., V.D., 6th August, 1926.

*To be Lieutenant (provisionally).*—Norman John Symington, 19th August, 1926; Lieutenant-Colonel H. V. P. Conrick, D.S.O., is brought on the authorized establishment of Lieutenant-Colonels, 18th August, 1926.

Lieutenant-Colonel R. W. W. Walsh, D.S.O., is appointed to command the 5th Field Ambulance, and to be supernumerary to the establishment of Lieutenant-Colonels with pay and allowances of Major, 1st September, 1926, *vice* Lieutenant-Colonel R. A. P. Waugh, who relinquishes the command on 31st August, 1926, and is transferred to the Unattached List, 1st September, 1926. Lieutenant-Colonel A. L. Buchanan is appointed to command the 14th Field Ambulance, and to be supernumerary to the establishment of Lieutenant-Colonels with pay and allowances of Major, 15th October, 1926, *vice* Lieutenant-Colonel C. H. E. Lawes, who relinquishes the command on 14th October, 1926. *To be Captain (provisionally).*—William Ivor Townsend Hotten, 1st September, 1926. The provisional appointment of Captain T. Hamilton is confirmed. Lieutenant-Colonel W. L. Kirkwood, O.B.E., is brought on the authorized establishment of Lieutenant-Colonels, 1st September, 1926.

*Unattached List.*

Captain M. R. Finlayson is transferred from the Unattached List, 4th Military District, 24th August, 1926.

*Australian Army Medical Corps Reserve.*

*To be Honorary Lieutenant.*—Allan Robert Stanley Vickers, 6th July, 1926. Captain L. St. V. Welch is transferred to the Australian Army Medical Corps Reserve, 1st Military District, 5th July, 1926; Honorary Lieutenant R. B. Job is retired, under the provisions of Australian Military Regulation 152 (1), 24th July, 1926.

Captain J. W. Hart is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 15th September, 1926.

Captains D. H. Bodycombe, M.C., S. M. Ware, and R. Fraser are transferred from the Australian Army Medical Corps Reserve, 3rd Military District, 10th September, 1926; Honorary Captain W. H. Ward is appointed to the Australian Army Medical Corps, 3rd Military District, 1st September, 1926.

**Third Military District.**

*Australian Army Medical Corps.*

Honorary Captain H. G. Mitchell is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally), 27th July, 1926; Captain H. B. Graham, D.S.O., M.C., is transferred to the Unattached List, 1st July, 1926.

*To be Captain (provisionally), Supernumerary to the Establishment pending Absorption.*—David Berman, 23rd August, 1926. *To be Lieutenant (provisionally) Supernumerary to the Establishment pending Absorption.*—David Zacharin, 23rd August, 1926.

Captain H. McLorinan is appointed from the Australian Army Medical Corps Reserve, 10th August, 1926; Honorary Captain W. H. Ward is appointed from the Australian Army Medical Corps Reserve, 2nd Military District, and to be Captain (provisionally), supernumerary to the establishment pending absorption, 1st September, 1926.

*Unattached List.*

Colonel T. P. Dunhill, C.M.G., Majors W. H. Summons and P. G. Dane and Captains W. G. H. Cuscaden and P. Lalor are transferred to the Australian Army Medical Corps Reserve, 1st September, 1926; Captain R. O. Douglas is transferred to the Australian Army Medical Corps Reserve and to be Major, 1st September, 1926.

*Australian Army Medical Corps Reserve.*

Captain R. G. Braham is transferred from the Australian Army Medical Corps Reserve, 5th Military District, 7th July, 1926.

Captain J. S. Yule, M.C., is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 19th August, 1926.

*To be Honorary Captain.*—Vincent Stanislaus Parer, 4th August, 1926. Honorary Captain C. F. Harkin is retired, under the provisions of Australian Military Regulation 152 (1), 16th July, 1926.

Captains D. H. Bodycombe, M.C., S. M. Ware and R. Fraser are transferred to the Australian Army Medical Corps Reserve, 2nd Military District, 10th September, 1926.

Captain A. H. Baldwin is transferred to the Australian Army Medical Corps Reserve, 1st Military District, 20th September, 1926. Honorary Lieutenant G. C. Nicholson is retired under the provisions of Australian Military Regulation 152 (1), 5th September, 1926.

**Fourth Military District.**

*Australian Army Medical Corps.*

Major R. L. Kenihan, M.C., is appointed to command the 6th Cavalry Field Ambulance, 1st September, 1926, *vice* Colonel M. H. Downey, D.S.O., who relinquishes the command on 31st August, 1926, and is transferred to the Unattached List, 1st September, 1926. Major H. P. Brownell, D.S.O., is appointed to command the 3rd Field Ambulance, 1st September, 1926, *vice* Lieutenant-Colonel E. A. H. Russell, who relinquishes the command on 31st August, 1926, and is transferred to the Unattached List, 1st September, 1926.

*Unattached List.*

Captain M. R. Finlayson is transferred to the Unattached List, 2nd Military District, 24th August, 1926; Colonel C. T. C. de Crespigny, D.S.O., is transferred to the Australian Army Medical Corps Reserve, 1st September, 1926.

*Australian Army Medical Corps Reserve.*

Major C. Bollen is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 29th July, 1926.

**Fifth Military District.**

*Thirteenth Mixed Brigade—Staff.*

Major C. Morlet, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Mixed Brigade Headquarters, 1st September, 1926, *vice* Major J. Bentley, M.C., who relinquishes the appointment on 31st August, 1926.

*District Base—Staff.*

Major J. Dale, O.B.E., Australian Army Medical Corps, is reappointed Assistant Director of Hygiene, District Base Headquarters, for a period of one year from 2nd September, 1926.

*Australian Army Medical Corps.*

Captain A. N. Jacobs, M.M., is brought on the authorized establishment, 1st July, 1926; Captain R. H. Crisp is transferred to the Australian Army Medical Corps Reserve, 30th June, 1926.

Honorary Captain H. L. Johnston is appointed from the Australian Army Medical Corps Reserve, and to be Captain (provisionally), 28th July, 1926.

Honorary Captain J. J. Hough and Honorary Lieutenant T. B. Seed are appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally) and Lieutenant (provisionally), respectively, and to be supernumerary to the establishment pending absorption, 28th July, 1926; Lieutenant-Colonel V. O. Stacy, O.B.E., is transferred to the Unattached List, 1st July, 1926.

Major J. Bentley, M.C., is appointed to command the 13th Field Ambulance, 1st September, 1926, *vice* Lieutenant-Colonel R. S. McGregor, D.S.O., who relinquishes the command on 31st August, 1926, and is transferred to the Unattached List, 1st September, 1926. Major C. Morlet, D.S.O., is transferred from the Unattached List, 1st September, 1926. Captain (provisionally) G. R. Troup is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st September, 1926. The age for retirement of Captain W. E. Blackall is extended for a period of one year from 8th July, 1926.

*Australian Army Medical Corps Reserve.*

Captain R. G. Braham is transferred to the Australian Army Medical Corps Reserve, 3rd Military District, 7th July, 1926.

**Sixth Military District.**

*Twelfth Mixed Brigade—Staff.*

Lieutenant-Colonel H. N. Butler, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Mixed Brigade Headquarters, with pay and allowances of Major, 1st September, 1926, *vice* Major C. G. Thompson, who relinquishes the appointment on 31st August, 1926.

*District Base—Staff.*

Captain (temporary Major) C. N. Atkins, Australian Army Medical Corps, is reappointed Assistant Director of Hygiene, District Base Headquarters, for a period of one year from 1st September, 1926.

*Australian Army Medical Corps.*

Lieutenant-Colonel W. L. Crowther, D.S.O., is appointed to command the 12th Field Ambulance, and to be supernumerary to the establishment of Lieutenant-Colonels, with pay and allowances of Major, 1st September, 1926, *vice* Lieutenant-Colonel H. N Butler, D.S.O., who relinquishes the command on 31st August, 1926.

*Australian Army Medical Corps Reserve.*

*To be Honorary Lieutenant.*—Matthew James Morris, 1st July, 1926.—(Ex. Min. No. 172.)

*To be Captain.*—Eugene Augustine Rogers, 18th August, 1926.

*Award of the Colonial Auxiliary Forces Officers' Decoration.*

*Australian Army Medical Corps.*—Lieutenant-Colonel J. A. Newell.

# Congress Notes.

## AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following programmes of sections have been issued by the Executive Committee of the second session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927.

SECTION IV.—PATHOLOGY AND BACTERIOLOGY.

*President:* Dr. A. H. Tebbutt, D.S.O.

*Vice-Presidents:* Dr. R. J. Bull, Dr. J. Dale, Dr. L. V. L. Duhig, Dr. A. B. Pearson.

*Honorary Secretary:* Professor A. M. Drennan.

*Friday, February 4, 1927.*

Morning.—Combined meeting with Sections I., II., V., XII.: "Goître."

"The Pathology of Goître," by Professor A. M. Drennan.

"Pathology of the Thyreoid Gland."

Afternoon.—Papers on goître, by Dr. A. H. Tebbutt, Dr. V. R. Woodhill and Dr. F. S. Hansman.

"Comparative Therapeutic Value of Thyreoid Extract," by Dr. F. G. Morgan.

*Monday, February 7, 1927.*

Morning.—Papers on renal and blood conditions:

"Paths of Renal Infection," by Dr. C. H. Kellaway.

"Renal Tumours," by Dr. E. W. Ferguson.

"Review of Hæmatology in New South Wales," by Dr. Elsie Dalyell.

"Some Considerations of the Anæmias, Leuchæmias and Allied Conditions," by Dr. L. B. Bull and Professor J. B. Cleland.

Afternoon.—"Poliomyelitis," by Dr. C. M. Hector.

"Specific Localization of Streptococci," by Dr. C. H. Kellaway.

"Methods of Obtaining Blood from Human Donors," by Dr. F. G. Morgan.

*Tuesday, February 8, 1927.*

Morning.—"The Value of Comparative Pathology in the Problems of Human Disease," by Dr. L. B. Bull (presented by Professor J. B. Cleland).

"Wassermann and Kahn Reactions," by Dr. E. D'Ath.

"Gonococcal Fixation," by Dr. Elsie Dalyell.

Afternoon.—Combined meeting with Sections II. and V. on parasitology and tropical conditions:

"Diagnostic Tests in Hydatid Disease," by Dr. C. H. Kellaway.

"Hookworm in Samoa," by Dr. T. R. Ritchie.

"Hookworm in Australia," by Dr. R. W. Cilento (or Dr. T. C. Backhouse).

"Yaws," by Dr. T. R. Ritchie and Dr. A. H. Baldwin.

*Wednesday, February 9, 1927.*

Morning (until 11.30 a.m.): "Differential Action of X Rays," by Dr. W. Moppett.

"Tuberculosis as Met with Post Mortem in Australia and Inferences to the Drawn Therefrom," by Professor J. B. Cleland.

(At 11.30 a.m.)—Meeting of all Sections: "The Present Position of Cancer," by Professor P. MacCallum.

Afternoon.—Combined meeting with Sections II. and XII.: "Inflammation and Tumours of Bone," by Dr. W. Keith Inglis.

SECTION VI.—OPHTHALMOLOGY.

*President:* Dr. A. M. Morgan.
*Vice-Presidents:* Dr. G. E. O. Fenwick, Dr. J. Lockhart Gibson, Dr. C. Gordon Macleod, Dr. D. D. Paton, Dr. J. F. Rudall.
*Honorary Secretary:* Dr. A. J. Hall.

*Friday, February 4, 1927.*

Morning.—President's Address: "Causes of Concomitant Strabismus," by Dr. A. M. Morgan.
"Ocular Signs of Ophthalmic Goitre," by Dr. Brook-Lewis.
"Surgery of the Lachrymal Sac," by Dr. W. Kent Hughes.

*Saturday, February 5, 1927.*

Morning.—Combined meeting with Section VII.: Diseases of the Eye and Disturbance of Vision Caused by Nasal and Sinus Affections. Opened by Dr. W. Kent Hughes: "Asthenopia and Nasal Insufficiency."

*Monday, February 7, 1927.*

Morning.—"Treatment of Non-Suppurative Intraocular Infections," by Dr. H. F. Shorney.
"The Uses of Red-Free Light in Ophthalmology," by Dr. J. Ringland Anderson.

*Tuesday, February 8, 1927.*

Morning.—"Treatment of Strabismus," by Dr. E. C. Temple Smith.
"Teaching of Ophthalmology for Medical Students," by Dr. Leonard J. C. Mitchell.

*Wednesday, February 9, 1927.*

Morning.—"Treatment of Optic Atrophy in Syphilis by Infection with Malaria," by Dr. Z. Schwartz.
"Retinitis Pigmentosa in a Family," by Dr. Leonard J. C. Mitchell and Dr. Cyril Dixon.
"Iontophoresis in Ophthalmic Work," by Dr. E. C. Temple Smith.

## JOHN IRVINE HUNTER MEMORIAL FUND.

THE following additional subscriptions have been received by the Honorary Treasurers of the John Irvine Hunter Memorial Fund:

| | £ | s. | d. |
|---|---|---|---|
| Previously acknowledged .. .. .. .. .. | 2,283 | 14 | 0 |
| The University Attendants' Association .. .. | 100 | 0 | 0 |
| Women Undergraduates (balance of subscription) .. .. .. .. .. .. .. .. .. | 2 | 9 | 2 |
| N. H. Fairley, Esquire .. .. .. .. .. .. | 5 | 5 | 0 |
| C. W. Witham, Esquire .. .. .. .. .. .. | 1 | 1 | 0 |
| | £2,392 | 9 | 2 |

## Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxvi.

COMMONWEALTH DEPARTMENT OF HEALTH, PORT ADELAIDE: Medical Officer.
COMMONWEALTH DEPARTMENT OF HEALTH, MELBOURNE: Medical Officer.
PEAK DOWNS HOSPITAL, CLERMONT, QUEENSLAND: Resident Surgeon.
QUEEN'S (MATERNITY) HOME, ROSE PARK, SOUTH AUSTRALIA: Resident House Surgeon.
RAILWAY CONSTRUCTION WORKS, NORTHERN TERRITORY: Medical Officer.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, SYDNEY: (1) Resident Medical Officer; (2) Junior Resident Medical Officer.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide. | Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia.<br>Yarloop Hospital Fund. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

## Diary for the Month.

DEC. 1.—Victorian Branch, B.M.A.: Annual General Meeting. Election of Council, 1927.
DEC. 1.—Western Australian Branch, B.M.A.: Council.
DEC. 2.—South Australian Branch, B.M.A.: Council.
DEC. 7.—Tasmanian Branch, B.M.A.: Council.
DEC. 7.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 8.—South Sydney Medical Association, New South Wales.
DEC. 9.—New South Wales Branch, B.M.A.: Branch.
DEC. 9.—Victorian Branch, B.M.A.: Council, Election of Office bearers, 1927.
DEC. 10.—Queensland Branch, B.M.A.: Branch (Annual).
DEC. 13.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 14.—Tasmanian Branch, B.M.A.: Branch.
DEC. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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